"I would have everie man write what he knowes and no more."—MONTAIGNE

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EDITORIAL

THE season at which an epidemic of anterior poliomyelitis may be expected will soon be at hand again.

We have no means of foretelling its severity, but we should clearly be wise to benefit from the experience of others. It is reassuring, therefore, to know that the Ministry of Health has been considering the implications of the epidemic in Denmark of which Lassen (1953) gave such a graphic description.

The outstanding feature of that outbreak was the large number of patients with bulbar involvement, who exhibited the triad of impairment of swallowing, pooling of secretions in the hypopharynx and respiratory insufficiency. During the worst two months of the epidemic patients of this type were being admitted at the rate of 20–30 a week to a hospital serving a population of little over a million, and between 40 and 70 patients at a time had to be sustained by artificial ventilation.

It is widely recognized that anaesthetists, especially those attached to thoracic units, can contribute usefully to the treatment of these cases. The matter may soon become urgent, and they should start thinking now about the issues involved.

The two main issues are: should mechanical respirators use positive or negative pressure; and should early tracheotomy be often or seldom performed.

The negative pressure respirators or "iron lungs" available in this country are nearly all of one type, which was designed many years ago. There are far too few of them to cope with an outbreak on anything like the scale of the Danish one. The decision as to whether negative pressure respirators have any place in the treatment of the respiratory insufficiency of poliomyelitis will have to be made. If we think they have, the design of existing machines will have to be changed, and a working party has, in fact, been set up by the Ministry of Health to produce an improved type of cabinet respirator. Some progress has been made by this body and by others working independently of it. It will be a long time, however, before well-designed cabinet respirators are available in any numbers. In the meanwhile the Ministry of Health has issued a memorandum (R.H.B. [53] 56) about modifications which can be made to existing machines. This is all very well as a stop-gap, but it will not appeal to those who have suffered from the basic faults in the design of the original machine (which is sometimes referred to abroad as "the English coffin"); and it seems clear that the only sensible short-term policy is to

press on with the development of a positive pressure machine. Such a machine should be cheap, simple and robust, and sufficiently reliable for one attendant to look after several.

The Danes, faced with a shortage of cabinet respirators during their epidemic, were forced to resort at first to hand power to keep their paralyzed patients alive, and two hundred medical students at once were thus employed. In a commendably short time, however, a fairly simple positive pressure machine was evolved (Bang, 1953) and was used with considerable success.

Several workers in this country have also devised positive pressure machines during the past year, and in the current Educational Supplement will be found a review of these and the principles involved. We ought now to make up our minds which machine is the best, and to start producing it at once, so that sufficient numbers will be available to cope with a large epidemic this summer.

Clinicians are still hesitant to advocate early tracheotomy in every patient with impairment of swallowing and respiratory insufficiency, largely from a fear of ulceration or of ultimate stenosis; but most people with experience of this form of treatment, which saved so many lives in Denmark, are convinced of its necessity. There is no doubt, too, that a patient with a tracheotomy can be handled far better with a positive pressure machine attached to the tracheotomy tube than in a cabinet respirator, in which the proximity of the neck steel to the stoma may cause serious difficulties.

Anaesthetists and other respiratory physiologists should think about these problems now, talk to their colleagues and define their attitude, so that if we have an epidemic here an agreed plan can be put into operation at once.

IN the Educational Supplement of this issue there will be found a description of the proposed new Department of Anaesthesia in the new building of the Royal College of Surgeons. The space which the Royal College has put at the disposal of the proposed new Department is adequate and, indeed, generous.

There is no Department of Anaesthesia in London which is staffed by full-time workers who are not responsible for maintenance of an anaesthetic service. When the Faculty of Anaesthetists institute a new Department they will be doing a service not only to London, inevitably the main centre of postgraduate teaching in this country, but also they will take a step which will materially assist British anaesthesia to maintain its undisputed preeminence in the world.

The Faculty will have to overcome many hurdles. Although it is not envisaged that the Head of the new Department will be concerned with undergraduate teaching, nevertheless he will have to be afforded clinical facilities, presumably in one of the principal central hospitals, and this, in itself, will take careful and tactful negotiation. The symbiosis between an academic department and a hospital can only be beneficial to both parties.

The main hurdle, however, which must be surmounted in the near future, if this project is to go forward, is to find the necessary finance. We believe this is the problem not only of the Board of the

Faculty of Anaesthetists, but also, by virtue of the national character of this Department, it should be a concern for every anaesthetist in this country. What can they do? It is clear that they may give individual donations, and the value of these may be nearly doubled by covenanting to give a regular donation for seven years, under which circumstances the Faculty could reclaim the tax paid by the donor. Any money likely to be raised in this way, although a handsome gesture and a very acceptable assistance, would not go far towards realising the £10,000 per annum which will be necessary to run the Department. For the main support, the Faculty must look to the large pharmaceutical firms which have a direct interest in anaesthetic research and to great industrialists who may be seeking a worthy outlet for philanthropic endeavour. One could reasonably put it to such possible donors that no other specialty in medicine has contributed so much directly to the relief of suffering and pain. They have only to picture the scene of any surgical operation only just over 100 years ago to appreciate this factor, and a little development of this theme would illustrate the absolute dependence on good anaesthesia of the phenomenal advances in surgery in recent times.

It is up to each and every practising anaesthetist in this country, and indeed those in the Commonwealth might like to join with their colleagues at home, to draw the attention of all with whom they are in contact, and who might be interested in this great project, to this need for funds. In this way, support will be on the widest possible basis and the new Department will be established as an academic and research centre worthy of British anaesthesia.

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BLEEDING FROM CUT SKIN AND SUBCUTANEOUS TISSUE SURFACES DURING CYCLOPROPANE ANAESTHESIA

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INTRODUCTION

ONE of the disadvantages of cyclopropane which has tended to bring the agent into disrepute, both among surgeons and anaesthetists, is the tendency for excessive bleeding to occur from cut skin and subcutaneous tissue surfaces during its administration. Many writers have commented upon this rather unfortunate property of an otherwise useful agent. Waters (1936) noted that cyclopropane relaxed peripheral vessels, while Marshall (1937), Boyd (1946) and Hewer (1948) all mentioned the excessive bleeding which occurred during its administration. Blair Gould (1949) stated that the excessive bleeding was confined to the skin and subcutaneous tissues only.

The tendency to bleed excessively during cyclopropane anaesthesia appears to vary in degree from patient to patient; to vary considerably in amount during the period of anaesthesia; and to be confined principally to the skin and subcutaneous tissue. There does seem to be some increase in bleeding from cut muscle surfaces, but from cut surfaces inside the abdominal cavity, whether visceral or peritoneal, increased bleeding is not the rule with this agent.

Foster, Neumann and Rovenstine (1945) using plethysmographic methods, demonstrated that cyclopropane, ether and thiopentone all produced peripheral vasodilatation which persisted throughout anaesthesia unless some factor such as blood loss, trauma, reflex stimulation, positional change, anoxia or deep anaesthesia was introduced.

Gillies (1950) stated that all anaesthetic agents produced peripheral vasodilatation.

Many others have reported similar findings and though any general anaesthetic agent, by bringing about vasodilatation appears likely to produce increased bleeding from operation wounds, it must be remembered that there are other factors which may also increase bleeding and which bear no relationship whatever to the particular anaesthetic used. Gillies (1950) gave a very comprehensive survey of the causation of excessive bleeding during surgical operations.

Included among non-anaesthetic factors are the following:

(i) Any pathological condition which prolongs bleeding time or coagulation-time.

(ii) Venous congestion secondary to cardiac or pulmonary disease.

(iii) Pyrexial conditions and diseases associated with raised basal metabolic rate.

(iv) Operations involving body tissues which are particularly vascular, such as, for example, muscle or glandular tissues.

(v) Increased intrapulmonary pressure, which may be due to a variety of causes, including (a) the resistance offered by the anaesthetic machine, and (b) respiratory obstruction produced by any cause. Bedford and Wright (1924), McNeal, Hutchison and McLaughlin (1948), and Price, King, Elder, Libien and Dripps (1951) all confirmed that any increase in intrapulmonary pressure was associated with a rise in venous pressure and this inevitably leads to increased bleeding from the operation wound.

(vi) Operations upon an unavoidably dependent site.

(vii) Increase in the carbon dioxide content of the blood. This may be brought about either by inefficient absorption during closed circuit anaesthesia, or, particularly in the case of cyclopropane, by retention secondary to depression of respiration by the anaesthetic agent. Barron (1946) stated that a rise in hydrogen ion concentration, or an increase in the carbon dioxide tension of the blood produced dilatation of the arterioles, while Wright (1952) reported that increase in either of these two factors produced dilatation of the arterioles and capillaries.

(viii) The improper use of controlled or aided respiration. Watrous, Davis and Anderson (1950), discussing the control of respiration in anaesthesia, pointed out that a continuous positive pressure of 15 mm. Hg caused an average rise in venous pressure of 41 per cent when the chest was allowed to expand.

Finally it is always thought that patients with a raised blood pressure tend to bleed more during operation than those whose pressure is within normal ranges. The systolic and diastolic blood pressures are scaled down as the blood passes through the larger arteries to the arterioles and thence to the capillary bed. The greatest fall has been shown to occur in the arterioles, but the capillaries also form part of the peripheral resistance and Landis (1930) demonstrated a definite pressure gradient from the arteriolar to the venular ends of the capillary loops. Ellis and Weiss (1930) estimated the pressure in the arterioles, capillaries and veins of the skin and found the mean pressure values in normal subjects to be: arterioles 55 mm. Hg, capillaries 9 mm. Hg, and veins 3 mm. Hg. In patients with hypertensive disease the mean arteriolar pressure was 108 mm. Hg, while the mean capillary pressure in these cases was 12 mm. Hg. The capillaries can normally resist the distending force of a raised arteriolar or venous pressure but during any form of general anaesthesia, peripheral dilatation which involves the capillary bed occurs, and under these conditions the pressure in the capillaries will follow passively any increase in pressure within the arterioles or veins.

This investigation was carried out (i) to determine the blood loss from a standard skin puncture in the various planes of stage 3 cyclopropane anaesthesia and to compare this with the blood loss under exactly similar circumstances during ether anaesthesia; and (ii) to ascertain whether there existed any correlation between the depth of cyclopropane anaesthesia and the blood loss from the skin wound.

Estimations of this loss were made before and during the administration of

cyclopropane in 100 cases, and similarly before and during ether anaesthesia in 50 cases. In 15 cases anaesthetized with cyclopropane the effects of hyperventilation through soda-lime upon blood loss were observed.

METHOD

To measure the blood loss, a standard puncture was made with an automatic lancet having a blade 3 mm. in length.

Ivy, Shapiro and Melnick (1935) and Ivy, Nelson and Bucher (1940) measuring bleeding-time, had noted the importance of capillary tonicity in aiding haemostasis by bringing about contraction of the capillary walls, and suggested, to overcome this factor, the application of a sphygmomanometer cuff round the arm and the maintenance therein of a pressure of 40 mm. Hg while the test was made.

This venostasis technique of Ivy was the one adopted in this investigation and the puncture was made into the skin of the dorsal surface of the finger below the base of the nail, immediately after the sphygmomanometer cuff had been inflated to 40 mm. Hg. The blood shed from this puncture was collected at intervals of 15 seconds on to filter paper, and the area so obtained measured in square centimetres. Care was taken not to touch the wound with the filter paper since any trauma or stimulation to the cut surface was likely to prolong bleeding.

The estimations of blood loss were made on the day before operation when the patient had normally been in hospital for at least 24 hours. It was thought desirable to do this after the patient had been resting in bed for some time, so that the extremities were warm and the peripheral circulation in a state of optimum vasodilatation. In each case a note was made of the blood pressure and the physical state of the patient; whether they were short or tall, obese or thin, etc.

During anaesthesia the estimations of blood loss were made at intervals of 15 minutes, and at the same time the patients' blood pressure, pulse rate and the depth of anaesthesia were noted.

TECHNIQUE OF ANAESTHESIA

Premedication.

All patients were given atropine sulphate gr. 1/100 (0.65 mg.) and morphine sulphate gr. 1/6 (10 mg.) one hour before induction of anaesthesia.

The drugs given as premedication produce some alteration in the circulation of the skin and subcutaneous tissues. McDowall (1938) and Goodman and Gilman (1940) stated that atropine sulphate given in therapeutic doses might produce dilatation of the cutaneous blood vessels. Clinically, this dilatation, which is by no means uncommon, appears to affect the blush area, but in susceptible patients it may well be that a more generalized vasodilatation occurs. According to Goodman and Gilman therapeutic doses of morphine sulphate might also produce dilatation of the skin vessels of the blush area.

The sum of the effects of these two drugs in any one patient was impossible to assess, and although there were probably some cases where a degree of generalized dilatation of skin vessels did occur, it was found impossible to make any allowance for it.

Type of Anaesthetic Machine.

In all cases a Coxeter-Mushin absorber fitted to a standard Boyle machine was used. Fresh soda-lime was used for each case.

Mode of Anaesthesia.

Anaesthesia was induced with intravenous thiopentone sodium and the maximum dose used in any one case was 0.5 g. In the first series, where cyclopropane was the anaesthetic, 500 ml. of oxygen and 500 ml. of cyclopropane per min. were then given until an adequate depth of anaesthesia was achieved, when the flow of cyclopropane was reduced to 50–100 ml. per min. for maintenance. The flow of oxygen was kept at 500 ml. per min. throughout, and when the flow of cyclopropane was reduced to a maintenance dose nitrous oxide was added to the mixture at the rate of 500 ml. per min. This dilution of the oxygen with nitrous oxide was advised by Waters (1945).

In the second series, when ether was the anaesthetic agent, a high flow of nitrous oxide (5 l. per min.) and oxygen (1,500 ml. per min.) was given after the induction dose of thiopentone with the absorber out of circuit and the expiratory valve open, and ether was added slowly from the bottle on the upper circuit of the Boyle machine. This was continued for exactly 5 mins. when the flow of nitrous oxide and oxygen was reduced to 500 ml. per min. each, the absorber put into circuit, the expiratory valve closed, and the administration of ether continued from the container in the Coxeter-Mushin unit. This particular technique for ether was found to give a smoother induction than when using the ether container on the Coxeter-Mushin unit from the commencement of anaesthesia.

Estimation of the Depth of Anaesthesia.

For ether the classification of the stages and planes of anaesthesia as given for this anaesthetic by Guedel (1937) was used.

For cyclopropane it was necessary to modify Guedel's classification. Several writers have indeed suggested that one can use his classification without modification, but Guedel himself (1940), discussing the signs of cyclopropane anaesthesia, said: "My chart of the signs and stages of anaesthesia published a number of years ago . . . will not do for cyclopropane." He suggested that the only three signs of depth available were respiration, colour, and the degree of abdominal relaxation.

To differentiate between plane I and plane II of stage 3, the dictum of Waters and Schmidt (1934) was accepted, that in cyclopropane anaesthesia oscillation of the eyeball is present in plane I and its disappearance coincides with entry into plane II.

It appeared to be impossible to distinguish with any degree of accuracy between plane III and plane IV and they were therefore considered as the composite plane III-IV.

Transition from plane II to plane III-IV anaesthesia was distinguished by increasing depression of respiration, enhanced muscular relaxation, and increasing depression of response to reflex stimulation.

TYPES OF OPERATION

In the early part of this investigation patients were chosen who were having a variety of operations, all of which were

suitable for cyclopropane anaesthesia. These included orthopaedic operations, herniorrhaphies and gynaecological perineal operations. Later it was thought wise to reduce possible variables by concentrating on patients having one type of operation. Further, in selecting suitable cases it was essential to choose patients who were undergoing operations of comparatively short duration since, for obvious reasons, it was undesirable that any factor of shock should complicate the investigation. The gynaecological cases were therefore chosen and all the later patients in the series were undergoing anterior colporrhaphy, amputation of the cervix and posterior colpoperineorrhaphy, which shall be referred to hereafter as the "Manchester" operation. Only patients who were in good health, and free from respiratory disease, from peripheral vascular disease, from gross heart disease or marked anaemia were included. Any patient who was pyrexial was excluded. If the patient coughed or gagged during induction, or if any suspicion of respiratory obstruction occurred during anaesthesia, no estimations of blood loss were made.

ESTIMATION OF BLOOD LOSS

In considering the operative blood loss it seemed likely that the amount lost in the various planes of stage 3 anaesthesia would be on the average proportional to the pre-operative blood loss for each particular patient. It also appeared likely that the variation in the results would vary with the size of the pre-operative blood loss, or in other words a person with a large pre-operative blood loss would tend to show more variation in a particular plane of anaesthesia than a person in whom the pre-operative blood loss was small.

Examination of the results suggests that this is true on the whole, and so it seemed better from the statistical point of view to consider not the actual blood loss in the various planes of anaesthesia, but the ratio of these losses to the pre-operative blood loss in each case. Thus all the values given for the various planes of anaesthesia have been obtained by calculating the ratio

$$\frac{\text{Blood loss in particular plane}}{\text{Pre-operative blood loss}}$$

for each patient, and these shall hereafter be referred to as the blood loss ratios. This, theoretically, may not be the best method but it has the merit of comparative simplicity. Other methods would involve similar assumptions and would be considerably more complicated in presentation without any great gain in precision. If the number of estimations of blood loss made in any plane of anaesthesia exceeded one, the average of the readings was taken and indication of this made by putting the appropriate numeral in brackets after the particular blood loss ratio shown in tables I or II.

RESULTS

A. *Cyclopropane anaesthesia.*

The pre-operative blood loss in the cyclopropane series (table I) showed a range of from 0.46 to 6.30 sq. cm. with a mean of 1.74 sq. cm. A record of the patients' colouring, height, approximate weight, etc., was kept but no correlation was found between any particular feature in the physical make-up and the amount of blood shed from the standard puncture either before or during operation.

TABLE I

Blood loss during cyclopropane anaesthesia

Sex	Age	Pre-operative blood loss in sq. cm.	Blood loss ratio			
			Plane I	Plane II	Plane III-IV A.R +	Plane III-IV A.R —
F	57	1.37	8.72	5.40		
F	44	0.83			1.31	3.04
F	44	1.53		3.73	1.9	
F	35	0.75		5.04 (2)		
F	43	1.38		3.96 (3)		
F	44	1.57		3.26		7.26
M	55	2.30		3.01		8.48
M	23	1.53		5.71 (2)	4.42	
F	44	1.46			2.11 (2)	5.90
M	47	0.59		5.42	2.30 (2)	
F	70	1.90		4.50	2.74 (2)	
F	52	0.72		2.06	1.52	5.19
F	50	1.30		2.70		3.92 (2)
M	16	1.42	6.30	4.70 (2)		
F	42	1.26	6.64	3.22 (2)		
F	56	3.10		2.97 (2)		
F	19	1.80	7.78 (2)			
M	36	1.10	7.45 (3)			
M	32	1.00	4.10	2.80 (2)		
M	55	2.10		1.67 (2)		
F	57	1.32	8.79	3.33 (2)		
M	24	0.70	6.50	4.43		
F	40	0.95	20.11		3.60 (2)	
F	40	1.25		4.48 (2)	2.48	
F	40	1.90	14.32 (2)		2.42	
M	29	1.55	6.71	5.10		
F	33	6.30		2.90 (2)	0.78	
M	22	1.55		4.45 (2)		
M	56	1.10		2.77 (3)		
M	44	0.89		1.90 (2)		26.52
F	54	0.92		3.10	1.90	
M	56	3.12	4.81	2.72	0.85	
F	47	1.63			2.12 (2)	12.09
F	57	2.37		6.40	3.32	7.70
F	53	1.37	11.42		1.59 (2)	
M	43	1.65	6.24	4.69 (2)		
F	40	1.31		3.55 (3)		
F	37	0.46		6.26		10.80 (2)
M	23	3.10	4.10	2.40 (2)		
F	60	1.33		3.53 (2)		
F	40	1.85		4.99	2.38	
F	47	1.30		4.51 (2)	2.12	
F	51	2.70		4.44 (2)		
F	51	2.50		4.66	1.60	
F	48	2.76		4.42 (3)		
F	46	1.70		5.00	2.70	
F	53	1.40		6.00	2.00	
F	38	0.75	6.95	4.40	2.20	
F	49	1.94		3.56 (2)		
F	48	1.50		4.73 (2)		
F	39	1.82		4.68	2.17	7.56
M	37	1.35	5.41		2.74	6.75
M	34	0.77		3.86	1.09	20.39

TABLE I—*Continued*

Sex	Age	Pre-operative blood loss in sq. cm.	Blood loss ratio			
			Plane I	Plane II	Plane III-IV A.R +	Plane III-IV A.R —
F	59	2.00		4.05 (2)		
F	46	3.24		5.50	3.85	
F	45	1.63			2.13 (2)	12.09
F	44	2.10		4.33 (2)		8.07
F	38	0.90			2.18 (2)	5.67
F	47	1.14		3.55 (3)		
F	41	1.37	4.89	2.83 (2)		
F	46	2.20		3.77 (2)		
F	53	3.50		2.80 (2)		
F	59	2.83		2.67 (2)		
F	48	1.73		4.54 (3)		
F	56	0.98		3.82	2.08	
F	39	1.10	10.45	3.00 (2)		
F	57	2.60		3.46	3.00	9.89
F	53	3.10		5.32 (2)		
F	46	2.10		4.43 (3)		
F	63	0.76		4.58 (2)		
F	57	1.14		3.28 (3)		
F	68	1.60		4.00 (2)		
F	53	3.50		2.70 (2)		
F	64	2.00		4.30 (2)		
F	46	1.73		4.00 (2)	1.86	
F	31	2.99			2.64 (2)	8.89
F	57	1.74		4.11 (3)		
F	40	2.90		3.77 (2)		
F	51	1.97			2.00	8.70
F	50	1.83		4.10 (3)		
F	49	0.78		5.14	2.30	
F	67	2.34		6.80 (2)		
F	53	1.44	10.30	4.90 (2)	2.18	
F	47	1.74		4.84	2.11	
F	52	1.21		2.42	1.43	6.93
F	36	1.92		3.80 (2)		10.12
F	38	0.75		4.07	1.98	10.40
F	39	2.53	8.66	2.84		
F	47	1.10		3.97	1.90	
F	46	1.87		5.04 (2)		
F	37	1.34		3.48	1.92	
F	46	1.47			1.20	7.80
F	50	2.73		4.90 (2)		
F	64	0.84	11.20	2.94	0.96	8.42
F	57	1.59		4.12 (2)		11.64
F	60	0.76	9.71 (2)		2.50	
F	53	1.82		4.08 (2)		
F	53	2.70		3.76	2.46	9.40
F	61	2.43		2.98 (2)		
F	44	0.69	7.67	3.81 (2)		

A.R. + = With assisted respiration.

A.R. — = Without assisted respiration.

TABLE II

Blood loss during ether anaesthesia

Sex	Age	Pre-operative blood loss in sq. cm.	Blood loss ratio	
			Plane II	Plane III
F	38	3.94	2.86	0.89(2)
F	48	1.04	6.44	1.54
F	60	0.68	2.88(2)	
F	61	6.8	2.61	0.82
F	59	1.45	3.52	12.25
F	61	3.24	4.53(2)	1.26
F	36	2.0	1.29(5)	
F	34	0.38	1.26(2)	4.66
F	41	0.46	2.30(3)	1.80
F	51	1.17	1.80(3)	
F	58	1.77	1.99(2)	
F	53	1.47	1.86	0.82
F	59	1.56	2.99(2)	
F	42	1.7	2.45(2)	
F	64	1.79	3.55(3)	
F	49	2.48	3.55(2)	1.81
F	43	0.57	2.14	1.56
F	47	2.66	1.24(3)	
F	60	2.70	1.85(2)	1.06
F	61	0.55	1.36(2)	6.22
F	48	0.48	1.37(2)	1.96
F	52	2.80	1.93(3)	
F	52	3.40	2.23(2)	
F	28	2.30	2.22(2)	
F	51	1.86	2.46(2)	
F	57	1.74	6.76(2)	
F	57	1.46	2.37(2)	
F	49	0.34	2.12(2)	
F	60	1.28	4.50	1.42(2)
F	37	1.83	2.76(2)	
F	34	2.16	1.21(2)	
F	52	1.53	1.93(3)	
F	53	0.99	3.69	1.31
F	61	3.20	1.59(2)	
F	54	2.30	2.48	0.96
F	63	1.31	1.45(2)	
F	35	4.07	2.17(2)	
F	47	2.90	1.48(2)	1.08
F	36	0.33	1.70(3)	
F	37	1.54	2.33(2)	1.04
F	62	1.94	5.41(2)	8.77
F	66	2.24	2.15(2)	
F	55	2.08	2.94(3)	
F	43	3.38	1.97(2)	5.36
M	23	4.00	1.24(4)	
F	48	2.20	3.08	2.33
F	58	1.64	2.95(2)	
F	61	1.14	3.93	1.54
F	49	0.94	2.37(2)	
F	64	1.78	3.19(2)	1.18

The mean blood loss ratio in plane I, stage 3 anaesthesia was 8.30 ± 0.73. Statistically this was highly significantly different from the pre-operative blood loss at unity, since the two means differed by more than twice the standard error of the difference of their means. (The standard error of the difference was calculated from the formula $\sqrt{(S.E._1)^2 + (S.E._2)^2}$ where $S.E._1$ and $S.E._2$ were the standard errors of the two observations under consideration.)

The mean blood loss ratio in plane II was 4.00 ± 0.11 and this was significantly different from (i) the pre-operative blood loss, and (ii) the mean blood loss ratio in plane I (fig. 1).

TABLE III

Cyclopropane

Plane of Stage 3	No. of cases	Mean blood loss ratio
I	24	8.30±0.73
II	85	4.00±0.11
III–IV A.R.+	44	2.16±0.11
III–IV A.R.−	26	9.37±0.95

A.R.+ = With aided respiration
A.R.− = Without aided respiration

In plane III–IV the mean blood loss ratio was 2.16 ± 0.11 when respiration was aided and 9.37 ± 0.95 when the respiratory depression was deliberately allowed to go unaided (table III). Both these results were significantly different from the pre-operative blood loss. In the same way they were significantly different from each other, and from the mean blood loss ratio in both plane I and plane II.

B. *After hyperventilation during cyclopropane anaesthesia.*

The results are given in tables V and VI.

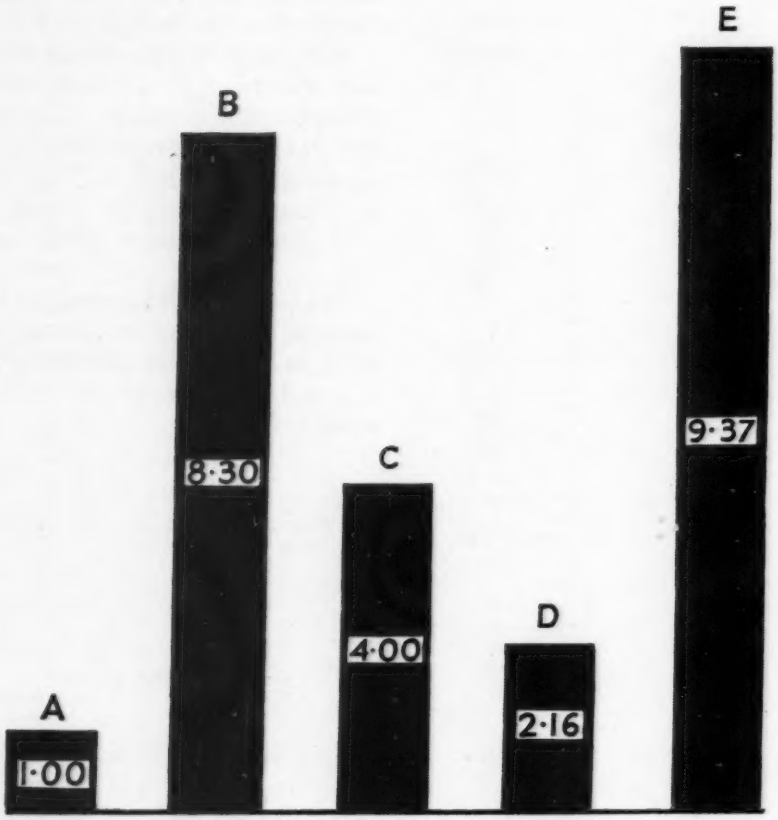

FIG. 1

To show the comparison between blood loss from the standard skin puncture in the various planes of stage 3 cyclopropane anaesthesia.

A—Mean pre-operative blood loss.
B—Mean blood loss ratio in plane I stage 3.
C—Mean blood loss ratio in plane II stage 3.
D—Mean blood loss ratio in plane III–IV with aided respiration.
E—Mean blood loss ratio in plane III–IV without aided respiration.

In eight cases in plane II, stage III of cyclopropane anaesthesia the mean blood loss ratio was reduced, after hyperventilation through soda-lime for five minutes, by 1.65 ± 0.48 (i.e. mean of column 4 – mean of column 6, table V) and this was equal to a mean reduction of 30.60 per cent.

In seven cases in plane III–IV, the mean reduction after hyperventilation amounted to 6.86 ± 2.40 or to 80.20 per cent.

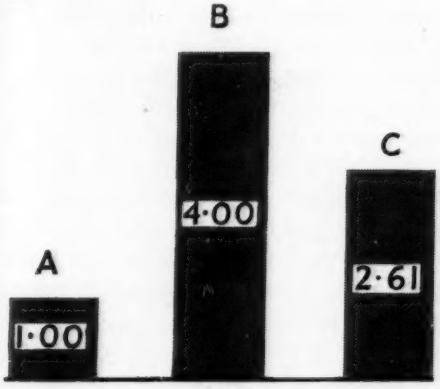

FIG. 2

To show the comparison between blood loss from the standard skin puncture in plane II stage 3 of cyclopropane and ether anaesthesia.

A—Mean pre-operative blood loss.
B—Mean blood loss ratio in plane II stage 3 cyclopropane anaesthesia (85 cases).
C—Mean blood loss ratio in plane II stage 3 ether anaesthesia (50 cases).

C. *Ether anaesthesia.*

The pre-operative blood loss in the ether series (table II) ranged from 0.33 to 6.80 sq. cm. with a mean of 1.92 sq. cm. As in the cyclopropane series no correlation existed between any feature in the physical make-up of these patients and the pre-operative or operative blood loss.

No estimations of blood loss were made in either plane I or plane IV, of stage 3.

TABLE IV

Ether

Plane	No. of cases	Mean blood loss ratio
II	50	2.61 ± 0.18
III	23	2.64 ± 0.61

TABLE V

Effect of hyperventilation through soda-lime upon blood loss. Plane II, stage 3—cyclopropane

Column 1—Sex
„ 2—Pre-operative blood loss
„ 3—Blood loss before hyperventilation
„ 4—Ratio increase
„ 5—Blood loss after hyperventilation
„ 6—Ratio increase on pre-operative blood loss
„ 7—Difference, i.e. Column 4—Column 6

1	2 (sq.cm.)	3 (sq.cm.)	4	5 (sq.cm.)	6	7
F	0.75	3.3	4.4	1.65	2.2	2.2
F	1.7	8.5	5.0	5.1	3.0	2.0
F	3.24	17.8	5.49	12.5	3.86	1.63
F	1.37	9.7	7.08	5.84	4.26	2.82
F	1.73	8.3	4.8	7.42	4.29	0.51
F	0.98	3.74	3.82	2.04	2.08	1.74
F	1.9	7.5	4.0	9.5	5.0	−1.0
F	1.37	11.95	8.72	7.4	5.40	3.32

TABLE VI

Effect of hyperventilation through soda-lime upon blood loss. Plane III–IV, stage 3—cyclopropane

Column 1—Sex
„ 2—Pre-operative blood loss
„ 3—Blood loss before hyperventilation
„ 4—Ratio increase
„ 5—Blood loss after hyperventilation
„ 6—Ratio increase on pre-operative blood loss
„ 7—Difference, i.e. Column 4—Column 6

1	2 (sq.cm.)	3 (sq.cm.)	4	5 (sq.cm.)	6	7
M	1.35	9.11	6.74	3.7	2.74	5.0
M	0.77	5.7	7.4	0.84	1.09	6.31
F	1.82	13.76	7.56	3.97	2.18	5.38
F	2.99	26.3	8.80	6.19	2.07	6.73
F	0.9	2.92	3.24	1.68	1.87	1.37
F	1.35	17.9	13.26	2.9	2.15	11.11
F	1.63	19.7	12.09	3.2	1.96	10.13

In plane II the mean blood loss ratio (table IV) was 2.61 ± 0.18, while in plane III the figure was 2.64 ± 0.61. Both these values were significantly different from the pre-operative blood loss, but not significantly different from each other. They were significantly different from the blood loss ratio in plane II of cyclopropane anaesthesia (fig. 2).

DISCUSSION

The bleeding produced by the infliction of the standard skin puncture comes from the capillary portion of the vascular tree, while the operation incision itself cuts small arteries and veins as well as capillaries. In this investigation it was presumed reasonable to take the amount of blood shed from the capillaries by the standard puncture as a quantitative and comparative indication of the bleeding from the skin vessels as a whole.

The anaesthetic agents in common use alter in varying degrees the dynamics of the peripheral circulation. These alterations arise from a combination of factors which include the potential properties of all agents to bring about (i) dilatation of the peripheral vessels (ii) alteration in cardiac output (iii) alteration in venous pressure, (iv) alteration in the pH and carbon dioxide content of the blood, and (v) alteration in blood pressure.

Robbins and Baxter (1938) in a study of the effects of cyclopropane anaesthesia upon the cardiac output in dogs, demonstrated that this was increased by as much as 45 per cent in plane II–III of stage 3, but in the deeper anaesthesia of plane III–IV the output showed a mean decrease of 28 per cent.

Prime and Gray (1952) in a study of cardiac output and forearm blood flow showed an immediate increase in both during cyclopropane anaesthesia, and the increases were maintained throughout anaesthesia in all but one case. Blalock (1927) experimenting upon dogs showed that during ether anaesthesia the cardiac output was increased from 7–100 per cent in all but three cases. In these three cases the depth of anaesthesia was profound. He concluded from his experiments that the lighter planes of ether anaesthesia increased cardiac output, and that deep anaesthesia produced a fall in output. Blalock suggested that the increase in cardiac output during light ether anaesthesia might be due in part to the increased respiratory effort which occurs in the early stages of anaesthesia with that agent, but since a similar increase occurs with cyclopropane where no respiratory stimulation manifests itself, this suggestion is unlikely.

Taking measurements of venous pressure during ether anaesthesia Meyer and Middleton (1930) found a pronounced rise during induction followed by a fall to a plateau somewhat elevated above the normal for the subject throughout the period of maintenance. They noted that reflex stimulation during light anaesthesia produced a temporary elevation of pressure.

Volpitto, Woodbury and Hamilton (1940) reported the occurrence of increased venous pressure during cyclopropane anaesthesia and they demonstrated very clearly the rise produced by increased intrapulmonary pressure.

Bennett, Bassett and Beecher (1944) experimenting upon dogs found a definite and progressive rise in venous pressure

during both cyclopropane and ether anaesthesia, but the rise was of smaller magnitude in the ether series.

Price, King, Elder, Libien and Dripps (1951) studied the effects of increased intrapulmonary pressure in man during cyclopropane anaesthesia and they were able to demonstrate an abrupt rise in venous pressure when such an increase occurred.

Price, Connor and Dripps (1953) demonstrated a rise in central venous pressure during cyclopropane anaesthesia. They found that while the increase was greatest in the presence of respiratory acidosis, it could occur in lesser degree, without accumulation of carbon dioxide.

Stormont, Hathaway, Shideman and Seevers (1942), and Dripps (1947) demonstrated varying degrees of carbon dioxide retention and acidosis during cyclopropane anaesthesia. This was produced by the depression of respiration which Waters (1936) was able to show manifested itself as early as plane I stage 3 of cyclopropane anaesthesia. In ether anaesthesia, on the other hand, depression of respiration does not occur earlier than plane III, and there is normally an increase in respiratory minute volume in planes I and II as stated earlier. Barron (1946) showed that a rise in hydrogen ion concentration or an increase in the carbon dioxide tension of the blood produced dilatation of the arterioles, while Wright (1952) reported that increase in either of the two factors was associated with dilatation of both arterioles and capillaries.

The blood pressure does not normally rise above the pre-operative value during cyclopropane anaesthesia. Waters (1945) reviewing 25,000 administrations of cyclopropane stated that in patients skilfully anaesthetized there should be no increase in blood pressure and the pulse rate should be slowed slightly to between sixty and seventy beats per minute. Should a rise of blood pressure occur, he considered that this was not due to the action of cyclopropane itself, but either to a build-up of carbon dioxide or to the presence of respiratory obstruction or oxygen lack. Further, a rise of blood pressure may occur from pressor reflexes arising from sensory stimulation in light anaesthesia.

It is interesting that Price, Connor and Dripps (1953) demonstrated in a recent work that increase in systolic blood pressure can occur without a rise in the carbon dioxide tension of the blood, but they also showed that the mean increase is always greater if a respiratory acidosis does in fact exist.

On the other hand, in light ether anaesthesia, there is normally a slight rise in blood pressure associated with an increased pulse rate.

The combination of some or all of these factors produce an increased peripheral blood flow during anaesthesia. Studies of such changes during cyclopropane anaesthesia were made by Abramson, Grollman and Schwartz (1941) with a plethysmographic method. In twelve normal subjects they found an increase in blood flow through the hand and forearm, but they were unable to show any correlation between the depth of anaesthesia and the magnitude of the increase in circulation. Lynn and Shackman (1951) using a plethysmograph measured circulatory changes in the limbs under several anaes-

thetic agents, including cyclopropane. With this anaesthetic they found an increase in limb flow which commenced immediately upon induction of anaesthesia, reached a maximum in between 15 and 30 minutes, and continued throughout the operation. They, too, were unable to find any correlation between the degree of increased blood flow in the limbs and the various planes of anaesthesia.

Shackman and Graber (1952) measured (i) the calf flow, and (ii) the skin temperatures of the great toe in the same or contralateral leg under thiopentone and cyclopropane anaesthesia, and found that in all the cases examined the skin temperature and the calf blood flow began to increase as soon as anaesthesia was induced and reached a maximum in 30 minutes. Prime and Gray (1952) demonstrated a similar increase in forearm blood flow during cyclopropane anaesthesia.

During ether anaesthesia Herrick, Essex and Baldes (1932) and Mann, Essex, Herrick and Baldes (1935) demonstrated increased blood flow through the peripheral arteries. The increases appeared to vary inversely with depth. Prime and Gray (1952) showed a marked increase in forearm blood flow during ether anaesthesia.

The results of the present investigation show that during cyclopropane and ether anaesthesia the increased blood flow brought about by varying combinations of the factors described above, was manifested by increased bleeding from the operation wound.

In cyclopropane anaesthesia this bleeding was greatly increased and appeared to be inversely proportional to depth in plane I and in plane II. These increases were to be expected since increased cardiac output and venous pressure was added to the already existing peripheral dilatation produced by the anaesthetic agent itself.

That bleeding was increased in plane II to a lesser degree than in plane I agrees with the dictum of Gillies cited earlier that the amount of bleeding during anaesthesia is inversely proportional to depth.

The experimental work of many workers has suggested that the increased cardiac output recorded by them in ether and cyclopropane anaesthesia was inversely proportional to the depth of anaesthesia, so that any reduction in bleeding in plane II as compared with plane I of cyclopropane anaesthesia can be accounted for by the combination of a reduced cardiac output, depression of the pressor reflex stimulation from the operative field and increasing depression of the vasomotor centre.

The results for plane III-IV were interesting. Table III shows that the blood loss ratio was 2.16 ± 0.11 when respiration was aided. In the majority of cases the surgeons reported less bleeding from the operative site than when the patients were in the lighter planes of anaesthesia. In plane III-IV when respiration was deliberately allowed to become depressed and was not aided the blood loss ratio rose to 9.37 ± 0.95. This increase may be accounted for in part by the anoxaemia which would accompany the depression of respiration, and which would itself produce some degree of peripheral vasodilatation, but the most important factor was almost certainly the increase in pH and carbon dioxide content of the blood conse-

quent upon diminished and inadequate respiration. To demonstrate if possible that this was so, the effect of hyperventilation for five minutes through soda-lime was estimated in some cases with the very striking results already noted.

Seevers, Stormont, Hathaway and Waters (1939) demonstrated the occurrence of a marked respiratory alkalosis following hyperventilation during anaesthesia, and Abramson and Ferris (1940) found a decrease in the blood flow through the hand following it. Kety and Schmidt (1946) found, very significantly, a fall in cardiac output following hyperventilation during anaesthesia.

In the ether series the mean blood loss ratio in plane II stage 3 was significantly different from that in the same plane of cyclopropane anaesthesia. It showed that bleeding in this plane was, on an average, rather more than half as great again when cyclopropane rather than ether was the agent used.

It seems reasonable to presume that bleeding in each plane of anaesthesia is therefore likely to be relatively greater when cyclopropane rather than ether is the agent. Certainly the mean blood loss ratio for cyclopropane in plane III-IV when respiration was not aided was greatly in excess of that for ether in plane III, and clinically, bleeding was much more marked from the skin wound in light cyclopropane than in light ether anaesthesia.

The mean blood loss ratio for ether in plane III stage 3 anaesthesia was much higher, when compared with that of plane II, than expected. Clinically, a reduction of vasomotor tone and skeletal muscle tone, together with a cardiac output at about, or sometimes below, normal in this plane, combine to produce a fall in blood pressure in ether anaesthesia and as a result bleeding tends to be reduced at this level.

The blood loss ratios in this plane were, with five exceptions, considerably below the mean blood loss ratio for plane II. If these five cases are excluded the remaining eighteen cases in plane III shown in table II have a mean blood loss ratio of 1.29 ± 0.08. This blood loss ratio is significantly different from the mean blood loss ratio for plane II anaesthesia. Examination of the records of the five cases with high blood loss ratio in this plane shows that their pre-operative blood losses at 1.45, 0.38, 0.55, 1.94 and 3.38 sq. cm. respectively were, with the possible exception of the last, well within the average for this investigation, and that the age of the patients did not appear to be a significant factor.

All the estimations of blood loss in plane III stage 3 of ether anaesthesia were made during the latter part of the Manchester operation when the posterior colpo-perineorrhaphy was being done, and at this part of the procedure reflex stimulation is possible in spite of the depth of anaesthesia. It is significant that while all the other cases showed a slight fall in blood pressure at this point, these five cases showed a slight fall in one only, maintenance at a steady level in another, and a rise in the remaining three cases. This suggests that the increased blood loss may have been mediated by cardiovascular reflexes from the operative field. On the other hand, it may well be that respiratory acidosis plays a part in producing the increased blood loss found in these cases,

but without data upon the pH and the carbon dioxide tension of the blood it is impossible to say whether or not this is so. It certainly occurs in the presence of respiratory depression, as many workers have shown and may well be a contributing factor here.

CONCLUSIONS

Though cyclopropane anaesthesia has been shown to produce more bleeding from skin wounds than ether, it is not possible and indeed it was not within the scope of this investigation to show why this should be so. The work of most investigators who have estimated cardiac output during the administration of both agents suggests that the increase in this factor was greater for any given plane of anaesthesia when cyclopropane rather than ether was used. Acting through a passively dilated peripheral vascular field such as has been shown to occur with both agents, this must be an important factor in producing the increased bleeding occurring in cyclopropane anaesthesia. Another important factor which appears to affect the amount of bleeding with this agent is the early onset of respiratory depression, with the consequent build-up of carbon dioxide and increase in blood pH. The marked differences in the blood loss ratio in plane III-IV when respiration was, or was not aided, and the substantial reduction in the blood loss ratio after hyperventilation support this view.

Whether one or both factors are among those responsible, the data obtained suggests that excessive bleeding from cut skin surfaces during cyclopropane anaesthesia can be reduced considerably in all cases, if a respiratory pattern possessing adequate tidal volume is maintained throughout anaesthesia and the lightness of plane I is avoided. Since the agent depresses respiration progressively as anaesthesia deepens and further since this depression commences as early as plane I of stage 3, satisfactory gaseous exchange with adequate removal of carbon dioxide can only be obtained by aiding respiration from the very commencement of anaesthesia.

SUMMARY

(1) An investigation was carried out to measure and compare the bleeding from standard skin punctures during cyclopropane and ether anaesthesia.

(2) The blood loss was estimated by measuring the area of blood collected upon a filter paper from this standard puncture, and the result was expressed in square centimetres.

(3) The mean pre-operative blood loss in 150 patients was 1.80 sq. cm.

(4) In plane I and plane II of cyclopropane anaesthesia the amount of bleeding was inversely proportional to depth. In the deeper anaesthesia of plane III-IV the blood loss was found to be dependent upon whether or not respiratory acidosis was prevented by aided respiration.

(5) Comparison of the mean blood loss ratios in plane II of cyclopropane and ether anaesthesia showed that, at this depth, cyclopropane produced approximately one and a half times as much bleeding as ether.

(6) Hyperventilation through soda-lime reduced the blood loss during cyclopropane anaesthesia.

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THE VENOUS RETURN

PHYSIOLOGICAL CONSIDERATIONS RELATED TO HEART FAILURE DURING ANAESTHESIA

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"CARDIAC RESUSCITATION" (Primrose, 1935) was the title of a paper published in 1935 based upon an experience of four successful cases. Other cases have, since then, been described in medical literature exhibiting features of interest along with suggestions as to the management of cardiac failure occurring in the course of surgical procedures. It is apparent, however, that in spite of the slowly accumulated experience of this condition, a group of cases of cardiac failure is becoming recognizable which resists treatment although a successful outcome is to be expected from the patient's general condition prior to the fatal incident. It is therefore proposed to re-examine the nature of the circulation, for it becomes clear that much more than the heart itself may be involved in the production of circulatory failure.

The recognition of a group of cases that resists the utmost efforts at cardiac massage leads to a distinction of cardiac failure into two definite types. There is the well-recognized failure of sudden onset, often of a temporary nature, usually occurring in the early stages of anaesthesia and due to any of many causes, supposed and real, acting upon the vasomotor control. Such cases, when the failure is complete, appear to be much more responsive to cardiac massage and constitute the majority of the successes. The second type consists of cases in which the failure is later in appearing, is slowly progressive and so may not be noticed until it is well advanced. In such cases, if the progressive deterioration cannot be checked, complete failure may ensue, and these cases may not respond to prolonged cardiac massage. In the course of more than fifteen years, four such cases have occurred in the writer's experience and, on reviewing these in retrospect, one feature stands out clearly and this is that the heart, pressed between the two hands, *could not be filled with blood*. It felt like a soft, round, pancake and never responded. In none of these cases was any pathological condition present which could conceivably be connected with circulatory failure. Postmortem findings in two of them showed the heart to be quite normal, and conditions of general healthiness had prevailed in them all. The anaesthetic, although blameworthy, cannot be a large factor in the fatal result when considered in relation to the century of experience of anaesthesia that has now passed; further, the same kind of failure may arise even with the most modern techniques.

Alexander and Hewer (1952) have

recently described a case of heart failure in which the resumption of cardiac activity seemed much less due to the use of 1 per cent procaine hydrochloride that was injected into the left ventricle than was the 1 ml. of liquid itself, the patient being an infant of 48 hours with a total heart capacity of 3–4 ml. They cite other literature in support of the view that the circulation can be greatly revived by injections of liquid, blood or saline.

It would appear, therefore, that the cause of heart failure is due to a defect in the venous return.

In the four cases mentioned, each with its full complement of blood, the fact did not occur at the time that much of the blood had become lost to the general circulation owing to excessive accommodation elsewhere. The capacity of the lower limbs, for instance, to accommodate blood is very considerable, this being well demonstrated in two renal cases in which the patient was positioned on the table in "knife-rest" inclination with legs dependent. After thirty minutes or so, the peripheral circulation had changed considerably and the pulse had become much weaker. This state was reported and, as the end of the operation was at hand, the restoration of the horizontal decubitus led to immediate improvement of the flagging circulation. Weakening or dilatation of the splanchnic area should not be lost sight of, as this area with its several blood depots can accommodate almost half the total blood content of the body. It has also been shown (Shackman, Graber and Melrose, 1952) that splanchnic dilatation occurs regularly as an observed fact following the initial venous dilatation seen in the early stages of a general anaesthetic.

In another way, this feature is recognized by Reid (1953) who states that intravenous drips should be started during the first few minutes of a general anaesthetic, when the superficial veins are dilated; for at the end, or after more than about fifteen or twenty minutes, the superficial veins may become so constricted that the procedure may be very difficult.

From these statements the delayed onset of the cases under consideration may be easily understood, as may the fact that certain subjects are more prone to the effects of this failure to adjust the circulation which, if not corrected in time, may proceed to a dangerous degree, for then the heart may receive insufficient blood for its propulsive efforts, and drugs given with the intention of stimulating this organ may fail. It must also be remembered that in the supine position, the heart is not well placed in a failing venous return, from whatever cause, as the blood is mostly lying at a lower level and has to be raised to this organ, and the power for doing so has largely disappeared. Failure of this kind does not appear to occur in cases maintained, often for several hours, in the prone position (some neurological operations). Before considering the power required for the return of blood to the heart, a few general remarks upon the haemal circulation may be helpful.

Circulation is an acknowledged feature of animal protoplasm, since all its chemistry depends upon it, and also many of its physical properties. Fundamentally, circulation means change of position of the liquid contents of an organism. While such movement in an organism is originally diffuse, it soon becomes directed into streams along the periphery of which enclosing endothelial tissue comes to be

laid down. With increasing organization such channels become recognized as clefts in the loose connective tissues of the body. The flat-worm Planaria shows this as a permanent condition; two simple endothelial tubes with a few small branches compose the entire vascular circulation which is activated by the general body movements. Streeter (1942) describes the same condition in the developing human embryo, the intra-embryonic channel on each side of the body exchanging its contents with those of the general coelom before vascular rudiments become effective as a circulation.

There is a still earlier circulation in which the body-stalk with the embryonic rudiment at its free end waves the latter about in the liquid of the blastocoele before any marked differentiation takes place. McLaren (1922) noticed this on removing early blastocysts from the uterine tubes of Cavia in his researches on the embedding of early blastocysts.

As organization proceeds, the watery mass of the developing embryo becomes replaced to an ever-increasing extent by connective tissues, the spaces of which are, according to Cajal (1933) lined by endothelium of mesenchymal origin; and although occupied by watery medium, this never exceeds the investing pellicle required for physiological exchanges. From the earliest stages of vascular development, the capillary portion of the system is permanently embedded in liquid medium while the rest of the tubular system is withdrawn from such body liquid by the laying down of impermeable fibro-elastic and muscular tissue in its walls. In this way, the fundamental principle of circulation—the movement of liquid within a liquid mass—is retained by the capillaries only, while the rest of the tubular system becomes engaged in maintaining this movement. Having thus briefly described the fundamental principle of circulation, one is in a better position to understand the operation of the two-sided nature of the human closed circulation.

Most attention has, up to the present, been devoted to the arterial side with its delicate adjustments of distribution; and there has been much measurement of its different pressures, in assessing the importance of blood pressure generally to the individual, especially under general anaesthesia. The recent development of hypotensive anaesthesia has, however, shown that vital processes can be carried on at pressures which are scarcely recordable. This fact alone seems to throw doubt on the ability of the heart to provide its own venous return which, nevertheless, is always adequate. The introduction of controlled respiration which is now so widely used, in association with artificially produced phrenic paralysis, also throws doubt on the value of negative intrathoracic pressure as assisting in the venous return, since this method is one of positive pressure breathing as opposed to the negative effect produced by the action of the diaphragm. Even in fracture of the cervical spine involving so much paralysis of the body musculature, the circulation is remarkably little disturbed, the venous return continuing without muscular help.

The commonly expressed idea that the *vis a tergo* of the ventricular systole is the sole important means of sustaining venous return, even with the secondary assistance of muscles and of breathing, must be reconsidered in view of the facts mentioned

above of anaesthesia and of paralysis. Is it to be believed that the capillary pressure can be the source of power for the return of blood to the heart when, in hypotensive anaesthesia, this pressure almost disappears? The problem thus comes to be the locating of the power that keeps the heart supplied with blood under all circumstances besetting its activity.

One has no hesitation in accepting the heart as a source of power, by virtue of its muscular walls, for the propulsion of blood to every part of the body, even if its power to return the same blood to itself appears to be inadequate. The completeness of its distributive function is unquestioned, but when it comes to the collection of blood from the entire capillary system and the delivery of it to the heart at the rate of, say, five litres per minute (Wright, 1952) a considerable step-up of energy is called for to achieve this.

Since the heart is a pressure pump only and does not suck as has often been supposed, the means for gathering the blood from the periphery and delivering it in volume to the heart for re-circulation calls for the application of mechanical principles, at or near the point where the venous return commences; it is here that the *vis a tergo* reaches its lowest value. The evidence of the existence of such mechanical means is provided by the histological researches of Spalteholz (1888) into the smaller units of the circulation. This authority states that in striped muscle, all the smaller veins and venules down to the smallest branches (0.25 mm.) are provided with valves allowing the blood to flow in the direction of the heart only. This statement, although made with reference to striped muscle only, is highly important, since such muscle constitutes by far the greatest mass of the body structure. From a statement by Krogh (1922) on the distribution of capillaries, the calculation is given for the total length of capillary tubing in man's muscles averaged at 50 kg., as 100,000 km. and their total surface as 6,300 sq. m. These unimaginable figures are mentioned merely to show that valved venules and veins are also present in very great numbers, a fact also asserted by Spalteholz.

The fact of the existence of valves in veins, particularly very small veins, does not seem to have attracted attention to their physiological value and importance, apart from the recognition that blood can only move in one direction. It is, however, necessary to recognize the existence and operation of a bio-mechanical principle which is exactly the same as that exhibited by the heart itself. Structurally this consists of a section of contractile tubing placed between two uni-directional valves. Functionally, the compression exerted by the contractile tube forces the blood against the peripheral valve which it closes, sending the blood through the proximal valve which it opens. When this principle is repeated several times in the course of a single small vessel and then repeated for every such vessel in the muscular mass, it will be realized that a very considerable accession of driving force is applied to the venous blood on its return to the heart. This force, built up as it is by innumerable individual efforts, shows itself as the steady flow characteristic of veins generally, the individual action always being a pulsatile contrac-

tion. This is a direct inference from the biomechanical principle involved, otherwise it could not work, any more than could the heart if it did not pulsate.

Looked at in this way, the return of blood from the muscles is energized by the minute efforts of innumerable venules and small veins, and their widespread and unorganized arrangement in the great muscle mass is the physiological complement of the highly-organized ventricles which set the blood off on the outward phase of their circuits by the succession of massive efforts.

A more pronounced instance of this mechanical principle is to be found in the portal system of veins. The veins draining the abdominal part of the alimentary tract are valved (Schafer, 1912) which means that the venous drainage is energized in the same way as is that of the body muscles. When it is realized that the mean pressure of the portal system of veins is almost double that of comparable body veins (Franklin, 1937), a very high degree of efficiency is reached which is, no doubt, correlated with the greater resistance of the hepatic capillary system and the heavier musculature of the portal vein itself.

In the case of the skin, Spalteholz (1893) in a later research points out that there are three venous plexuses: one, the sub-papillary which is sub-epithelial and has very small meshes; and two at different levels of the cutis which have larger meshes. It is in the third or deepest of these plexuses that valves are found, but *nowhere else* so far as the skin is concerned.

This observation is of importance since it shows that the driving force of the venous return from the skin is adequately effected by a single range of valved venules and indicates that valves are not laid down in venules indiscriminately, but are related to the nature and amount of function performed by the part.

Much the same arrangement is clearly present in the lymphatic drainage of the gut. The mesenteric and gastric lacteals which commence as closed sacs, and so have nothing comparable with the *vis a tergo* of the blood capillaries, are plentifully valved, giving them their beaded appearance which is their visible characteristic. Elsewhere, lymphatics are valved in proportion to the resistance offered to the lymph flow by the lymph glands which are normally packed with lymphocytes. Where this resistance is considerable, valves are found at much shorter intervals than is the case with venules (Schafer, 1912).

This diversion into the lymphatic system is important as showing the arrangements required to meet resistance. While the wall of the lymphatic is very distensible as compared with that of veins, distension cannot be allowed to any extent as this interferes with the efficient action of the muscular wall, and it is to prevent this that valves must be closely set in these channels. Compared with venules or small veins, the lymphatics have to do much more work in propelling their contents through chains of glands, after which passage the lymph flow is at very low pressure as it enters the venous system. This is indicated by the need for a single non-return valve on the venous pattern at the junction with the neck veins. The bio-mechanics of the lymphatic system are therefore of a most precise

nature, perfectly adapted to the special conditions under which they have to operate.

The lymphatic system of the gut possesses the very great advantage that the process of propulsion is almost visible, and it is entirely separated from any other form of vascular drive. This histological fact by itself leads one to doubt the importance attached to ventricular effort as being the principal motive power for the venous return.

The need for such a specialized mechanism for returning blood from the muscles as has been indicated arises from the peculiar nature of their function. When skeletal muscle contracts, it does so suddenly and powerfully, which fact calls for a rapid increase in blood supply. This latter has to be drained off with equal vigour and certainty from the contractile units. These are relieved of their blood by the very efficient venular system in the first instance, and to this there comes to be added the "muscle pump" action and the contractility of the larger veins themselves. When muscles are paralyzed or completely relaxed, it is evident that the venous return is entirely effected by the venular mechanism, which is the primary source of power for this purpose and which operates continuously irrespective of other circumstances. In lesser degree, the drainage of the skin and gut call for reduced ranges of valved venules as has been pointed out. This expresses the fluctuations of function exhibited by these organ systems. The skin, operating without fluctuation of any magnitude, has only a fringe of valves confined to one venous plexus; the gut, varying much more in its periodic functions is much better equipped with valve veins, while voluntary muscle excels all other structures in this respect on account of its extremely variable function.

The presence of valves in the larger veins of the body, particularly of the extremities, indicates a larger scale repetition of the venular function, and in these, a pulse has been observed synchronous with, but independent of, the arterial pulse which is not normally conducted across the capillaries and which is also centripetal (Franklin, 1937). These larger veins have to compensate for the effect of gravity which reaches a considerable value in man in the upright posture, and so muscular coat and valvular equipment are greater in the lower limb than the upper. Valves are not found in the trunk or head veins.

The solid viscera do not possess valved veins, a fact associated with uniformity of function and of blood demand as compared with the muscles, yet there are contractile elements overlying the capillary walls in all situations that perform circulatory function. Krogh (1922), after Vimtrup (1922), describes the contractile elements of the capillary walls known as Rouget cells, the thread-like processes of which surround the endothelial wall transversely. The contraction of these cells has been observed to start in the perinuclear protoplasm, and to spread along its filaments thus constricting the capillary wall locally. Movement of the blood is in the direction of the veins because the capillary effort is normally made against the arteriolar pressure behind. It is when this arteriolar pressure fails, as in death, that the movement of the blood becomes irregular or oscillatory before finally coming to a stop. This shows that the mechanics

of this system do not depend upon valves, as in the case of the venules and veins, but upon the back pressure offered by the arterioles. The Rouget cell phenomenon has not been universally accepted, contractility also being a better-known activity of the endothelial wall of the capillary. The latter, however, appears to be less rhythmical than that of the Rouget cell confirmed by Vimtrup.

Salisbury and Melvin (1936 a and b) by ophthalmic observation have given a clear idea of what occurs in the valveless veins of the retina, when the circulation fails as in approaching death. They show that the movement of the blood in the retinal vessels becomes irregular and the blood "lumpy" although continuous. Later, fragmentation definitely occurs, but there is still movement. The heart has stopped, but there is still good hope of resuscitation. This oscillatory movement lasts about ten minutes. When this stops, there is no hope of resuscitation.

This clearly observed fact of oscillation gives some idea of the rate of pulsation in these small veins, a rate that is possibly increased by the threat of death which circulation is attempting to prevent. It also shows that the arterial side of the circulation has been emptied by the venules since the heart stopped, and they can now no longer move the blood within themselves. It is this venular function persisting for a short time after death that has led the early anatomists, Aristotle among them, to assume that the arteries conveyed air to the various organs, a belief that lasted almost two thousand years.

Further evidence of this venous activity is given by Riml (1929). This experimenter arrested the pulmonary artery flow in a rabbit. The superior vena caval pressure rose at once approximately to 10 cm. of water to fall more slowly to 2–4 cm. The arterial pressure fell as the venous pressure rose. The blood in the great veins was collected at a pressure of 0.5–1 cm. of water, and was 50 per cent of the body total. As this was too much to come from the arterial side, Riml postulated the active contraction of veins and emptying of blood depots through the nervous system into the great veins.

This finding is demonstrated clinically in cases of "overdose apnoea" (Primrose, 1945).

This brief consideration of venular function has disclosed the existence of definite propulsive mechanisms entirely separate from that of the heart, and which differ from it fundamentally. Whereas, the feature of the ventricular effort is that of producing the high pressure required to effect the distribution of arterial blood, the function of the venular mechanism is to apply driving force at innumerable points on the periphery to set the blood in motion towards the heart. Venular function does little to restore pressure in the returning blood: it returns blood volume, the energy being spent on the gathering of the blood against the resistance offered by the constant reduction of the cross-sectional area of the venous system as the greater vessels are approached.

In this mobilizing of blood by the venules, the idea of pressure as a physical property, as exhibited by the arterial system, does not apply so readily to the venous channels. Pressure readings have been carefully recorded by many experimenters, even for intrathoracic veins in

which the pressure falls to values below the atmospheric level. Such results, although variable, have never been questioned, but it is clear that, physically, a liquid completely filling a container cannot exhibit a negative pressure. It, at least, always has its own weight and this cannot be reduced by any process of rarefaction as can a gas so as to give a pressure below some fixed standard. What is taking place is not negative pressure as such, for this would mean empty veins, but change of momentum that must occur with the fluctuating flow set up by the heart, as it accepts or refuses the blood offered to it by the veins. So long as there is blood in the veins, even with the low pressures given for, say, the external jugulars, they show no sign of being compressed by the atmospheric pressure on the outside which is uncompensated within the neck, or even on surgical exposure which removes fascial attachments said to prevent collapse of these vessels. While pressure may be the proper means of indicating the power of arterial distribution, which commences with a high positive value and which never becomes negative, it does not seem to be the correct means of determining the strength of venous activity, which is developed in quite another manner and in which the factor of pressure is definitely secondary to that of mass in motion, a fact also indicated by the different histology of the respective vessel walls. Rankin (1952) is in agreement with the principle stated concerning liquid completely filling a container such as the vascular system. This is not influenced by external conditions such as variations in thoracic pressure. The negative pressures recorded for intrathoracic veins are an expression of a relationship between velocity and pressure. If the former rises rapidly, the latter falls correspondingly according to certain formulae, but the effect is strictly local and of very short duration, playing little or no part in the act of filling the heart. Franklin (1937) discusses this feature of venous pressure and shows that veins of the larger sizes can only exert "tone" and never tension (that is, pressure). For the latter, valves are required and a resistance overcome. Valveless veins such as the inferior vena cava can exert tone in a variable degree, and when this is lost, its accommodation of blood may reach such proportions that the heart cannot be supplied with blood, a danger that occasionally appears during anaesthesia.

As the blood approaches the heart, its movement becomes "tidal" in the sense that a river on approaching the sea becomes tidal. This, due to cardiac activity, expresses the change of momentum to which the blood is subjected and it is the function of the caval veins to accommodate these disturbances by their variable tone.

Much has been spoken and written about the influence of respiration on the all-important function of heart-filling. The principal feature is that inspiration sets up the negative pressure in the chest that encourages the blood to occupy the great veins and atria and is therefore essential for the maintenance of the cardiac supply. This seems a doubtful theory especially when looked at from the point of view of overdose apnoea such as may be met with in the course of anaesthetic practice. This condition has been

described elsewhere (Primrose, 1945), but the points which concern the present discussion are the following.

As overdose approaches in a patient well supplied with oxygen, say, with chloroform (which, of all agents, shows this feature best), the medullary functions become depressed to the point at which breathing stops, as a defensive measure, preventing further intake of the drug and further depression of the circulation. This apnoea may last for several minutes while internal adjustments are being made which, in all cases, lead to spontaneous resumption of breathing. During this time, the depressed heart steadily increases in power, ultimately far exceeding the normal for general anaesthesia in those cases where the apnoea is prolonged to five or six minutes. In such a situation, there has been no breathing at all to create the negative pressure held to be so necessary for cardiac filling. The chest is always at rest in the expiratory position in which a negative intrathoracic pressure is absent. Not only, therefore, has the heart built up the effort to maintain the circulation in safety, but it has also carried its effort under natural stimulation far in excess of its normal for the state of general anaesthesia, a condition that is quickly relieved by the onset of breathing and return to normal filling. In these cases, which are so easily demonstrated, neither muscles nor respiration have played any part in keeping the heart supplied with blood. It is also to be noted that intra-thoracic negative pressure occurs some fifteen times a minute, whereas negative pressure in the large thoracic veins occurs some sixty or more times a minute, so that many auricular diastoles are unaccompanied by negative intra-thoracic pressure. Likewise, it matters little whether breathing is by natural inspiration with its negative pressure, or by forced inspiration with its positive pressure as in controlled respiration; the circulation is unaffected. There is, therefore, no relation of any physiological consequence between respiratory and circulatory fluctuations of pressure. Surgery and experiment also show that the heart keeps well filled even when the thorax (on one side) and the abdomen are both opened.

Venules and veins thus remain the only source of motive power for supplying blood to the heart, and until the enfeebled heart in an apnoea can regain its normal power and output, it may be actually embarrassed by the amount of blood offered by the veins. This fact, indicated by distended veins, is of importance as showing that the heart will not be allowed to fail so long as blood can be presented to it by the veins which, as already mentioned, continue their efforts for a short time after the heart has stopped. In cardiac resuscitation carried out on the human subject, this "short time" is narrowed down to about three minutes as the cortical cells cannot usually withstand longer anoxia and recover. Borderline cases show marked cerebral irritation which is a hopeful sign of ultimate recovery.

The effect of gravity on the circulating fluids of the body appears to be very slight in health. The exchange of fluid between the arteriolar and venular sides of the capillaries is always balanced, and even in the connective tissue spaces, only a pellicle of liquid bathes their walls. So soon, however, as the circulatory function falls

below a certain value, this fine balance is upset and oedema makes its appearance, and fluid at rest then comes under the influence of gravity.

To gravity is ascribed most of the cause of acquired varix of the lower extremities; but, strangely enough, the condition does not commonly arise where gravity would have most effect, namely, about the ankles and feet. The vessels here may remain normal with absence of oedema, unlike the condition presented by cardiac insufficiency in which oedema of the feet is a characteristic. The altered histology shows that the superficial veins become subjected locally to resistances which they endeavour to overcome. When hypertrophy fails, oedema does not occur so much as a vitiated venous circulation which may result in ulceration from the recirculation of venous blood locally, a condition that the valved arrangements make possible. Gravity rather makes the condition much more apparent to the sufferer from increased discomfort which is amenable to postural relief, than acts as a cause of varix.

In the healthy, the effect of gravity is easily accommodated. Acrobats and others practising the inverted position soon cease to be affected by it, even although there are no valves in the veins above the superior vena cava. These people do not suffer from congestion of the cephalic region.

The prime importance suggested for the venular system in returning blood to the heart is further accentuated by the fact that the terminal caval veins actually initiate the cardiac impulse in the sinus region which has its own particular rhythm. In fact, cardiac muscle with its special feature of spontaneous rhythmical contraction makes its appearance in the terminal caval veins, the amounts differing in different animals. In man (Lower, 1669), the superior vena cava alone is invested for part of its course by cardiac muscle and, according to Husten (1926), this reaches two-thirds of the way from the heart to the azygos vein entry. Not only is the venular system the prime mover of blood from the periphery to the heart under spontaneous action controlled by the sympathetic, but, at the central end, veins are the seat of instigation of the cardiac impulse, the heart being nothing more than an exaggerated vein with its great muscular walls situated between two sets of non-return valves.

Having now recognized the two power components of the vascular circulation with their mechanical and physiological differences, it is not remarkable that each should show its particular type of depression or failure. That of the ventricular side of the circulation has been dealt with in a previous communication (Primrose, 1935), in which it was shown that amyl nitrite is a drug of the utmost value since it gives an immediate diagnosis between temporary and permanent failure. In the former case, the recovery is immediate, while in the latter cardiac massage is the only treatment likely to initiate cardiac activity, for in these cases, the venous return can still supply the heart for a few minutes. Depression of the peripheral circulation such as may occur in high spinal and epidural anaesthesias has been closely studied by Evans (1944) who, with his adrenaline drip, has applied the physiological fact of the high susceptibility of the venular system to adrenaline. By this means, the venous return is main-

tained with sufficient energy to keep up a steady blood pressure on the arterial side. This can be maintained as long as required and varied as desired. Other drugs may now be used for this purpose with similar effect.

The success of such treatment for depression of the peripheral and venous circulation would seem to give strong support to the view of Henderson (1909) that capillary and venous depression are more important factors in the cause of shock than is vaso-motor depression, which is more likely to be sudden in its effects. In treating either of these conditions in which the effect of gravity may quickly make its appearance owing to lowered vitality, the legs should always be raised to a good height as they can contain a volume of blood that is urgently required elsewhere.

Such, then, is the view suggested for the venous circulation and its relation to cardiac filling, based upon the observation of certain features met with in anaesthetic practice and also upon well-established histological facts. These facts have lain dormant for more than half a century, but when brought to light, reveal the existence of a vascular mechanism of the first order. If it be permissible to invent a name for this mechanism, an appropriate one would be "The Venular Pump" which acts continually throughout the life of the individual, and even, for a short time, after cardiac arrest in the forlorn hope of saving life.

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CONCENTRATIONS OF DIETHYL ETHER IN THE BLOOD OF INTUBATED AND NON-INTUBATED PATIENTS

BY

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MANY papers have been published dealing with new agents, but less attention is paid to the great variety of ways in which established agents can be used. For example, there are many ways in which nitrous oxide, oxygen and diethyl ether could be employed for a short operation in which no marked abdominal relaxation is required. Two methods offer considerable contrast.

(*a*) To induce anaesthesia smoothly and as expeditiously as possible using nitrous oxide, oxygen and ether alone, and thereafter to maintain the level as light as possible but avoiding suboxygenation and making reasonably sure that vomiting, coughing, gagging, etc. do not occur.

(*b*) To induce anaesthesia smoothly and as quickly as possible using carbon dioxide as a respiratory stimulant until an oral cuffed endotracheal tube can be passed under direct vision, and to allow anaesthesia to lighten as much as is then possible when there is no risk of laryngeal spasm and there is security against aspiration, should vomiting occur.

In a comparison of these two methods the following questions arise. Will a much deeper level of anaesthesia be needed to pass the tube than is required for the maintenance of anaesthesia without a tube and, if this is so, will the deeper anaesthesia be enduring or transient? Given the security of the airway which a cuffed tube confers, will it in fact be possible to allow anaesthesia to become significantly lighter than when vomiting, coughing and spasm must be considered? Will the average degree of saturation of the patient with ether be different in the two cases? Will the concentrations of ether in the blood at the end of operation differ?

In an attempt to find the answers to these questions, the following investigation was carried out on unselected patients undergoing minor operations, requiring only light anaesthesia.

Arterialized venous blood samples were taken from the back of the warmed hand at approximately five-minute intervals. Blood ether concentration was estimated by a modification of Cavett's method (Knox and Pask, 1950).

The patients were divided into two broad groups:

Unsedated patients, premedicated with atropine only.

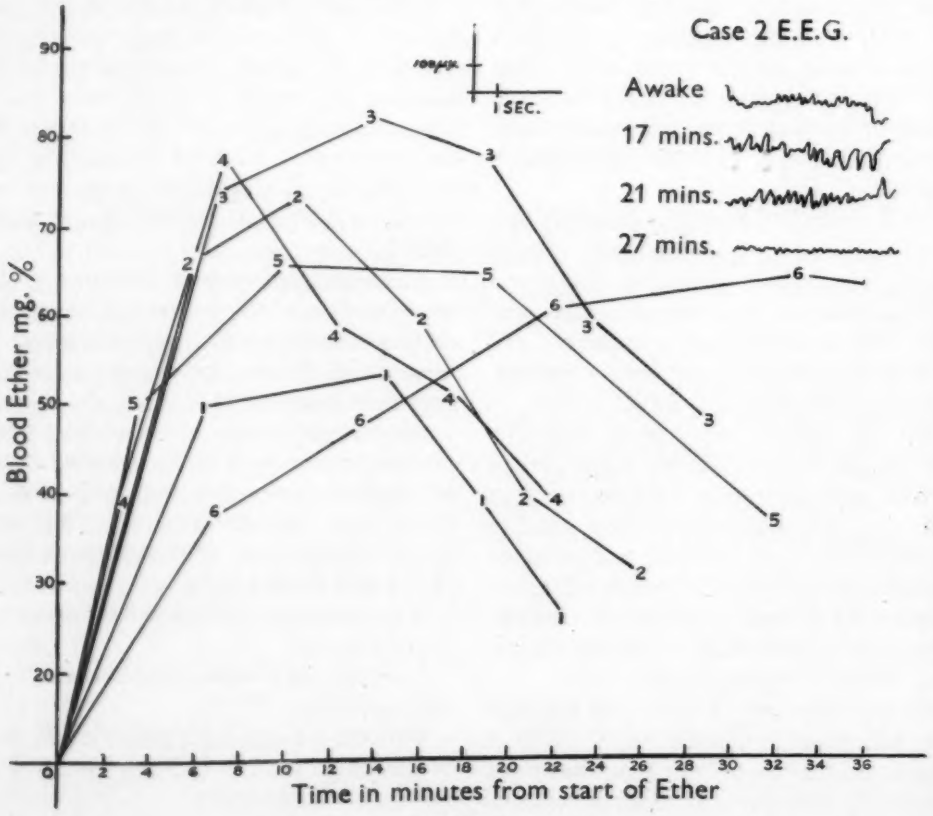
Patients sedated with pethidine 50 mg. or morphine gr. 1/8 (7.5 mg.) to gr. 1/6 (10 mg.) with atropine.

In one half of each group, induction

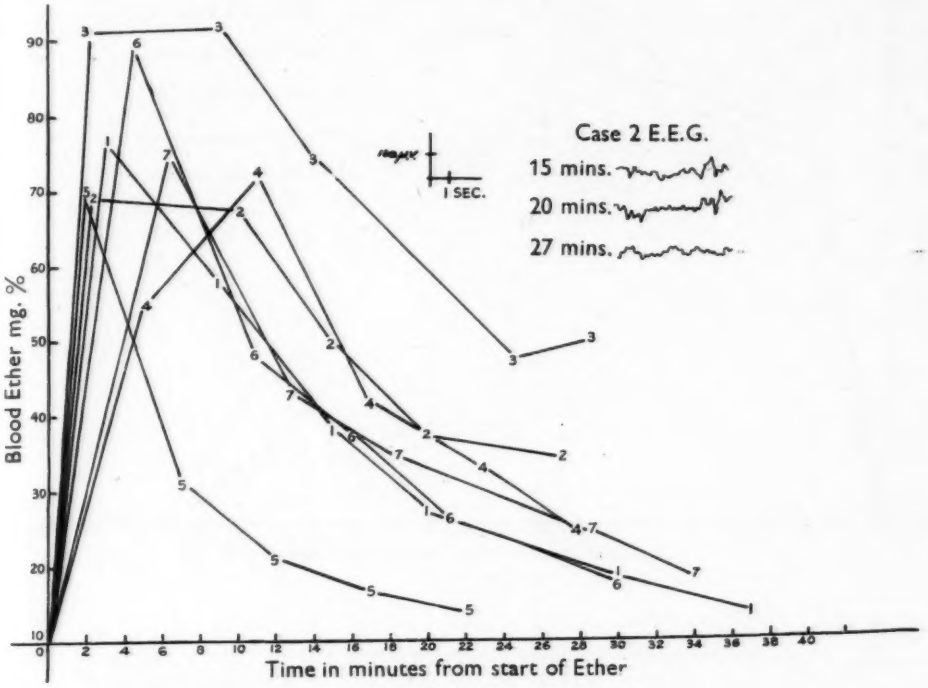
was carried out smoothly and as rapidly as possible without the aid of carbon dioxide until the patient entered stage III(i) (Guedel) when the first sample was taken.

In the other half of each group, induction was by nitrous oxide, oxygen and ether using liberal carbon dioxide to allow rapid deepening with ether. As soon as the jaw was relaxed, oral intubation was performed with the aid of a Macintosh laryngoscope, using a cuffed tube with non-analgesic lubricant. The first blood sample was taken at the time of intubation. In the majority of these cases, the ether could be turned off for long periods following intubation, maintenance being carried out with nitrous oxide 8 l./min. and oxygen 2 l./min.

In all cases, maintenance of anaesthesia was at the lightest possible level and, where possible, electroencephalographic recordings were made to confirm the clinical assessment of depth. Specimen traces are shown. The correlation between blood ether levels and E.E.G. recordings tallied closely with the findings of Faulconer (1952).



GRAPH I



GRAPH II

Graph I. Group 'A'. Six cases unsedated, not intubated. These followed a fairly consistent pattern.

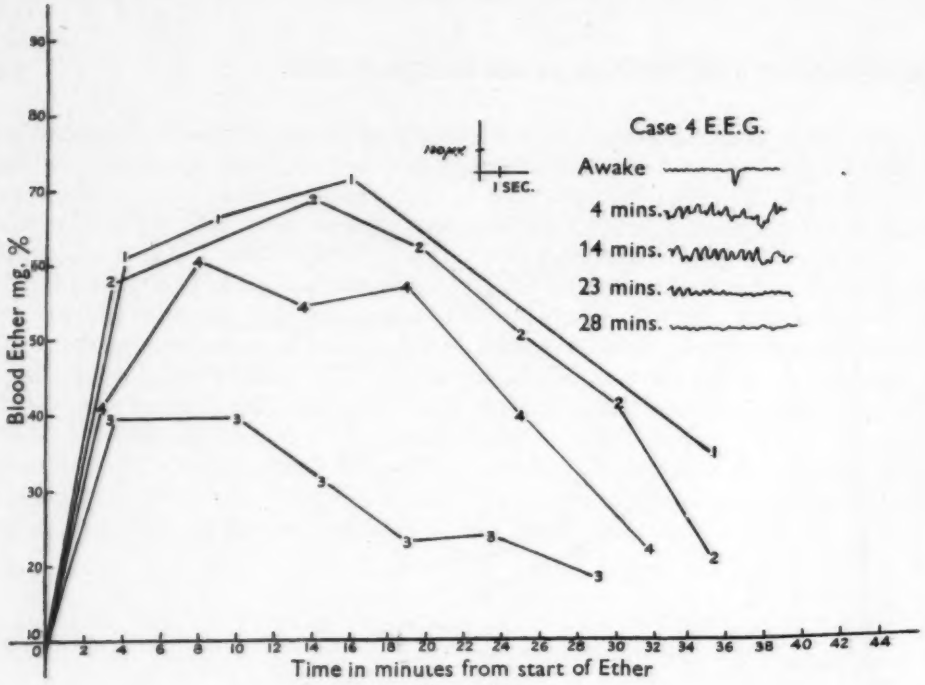
Graph II. Group 'B'. Seven cases unsedated, intubated and all consistent in pattern.

Graph III. Group 'C'. Four cases lightly sedated but not intubated and consistent in pattern.

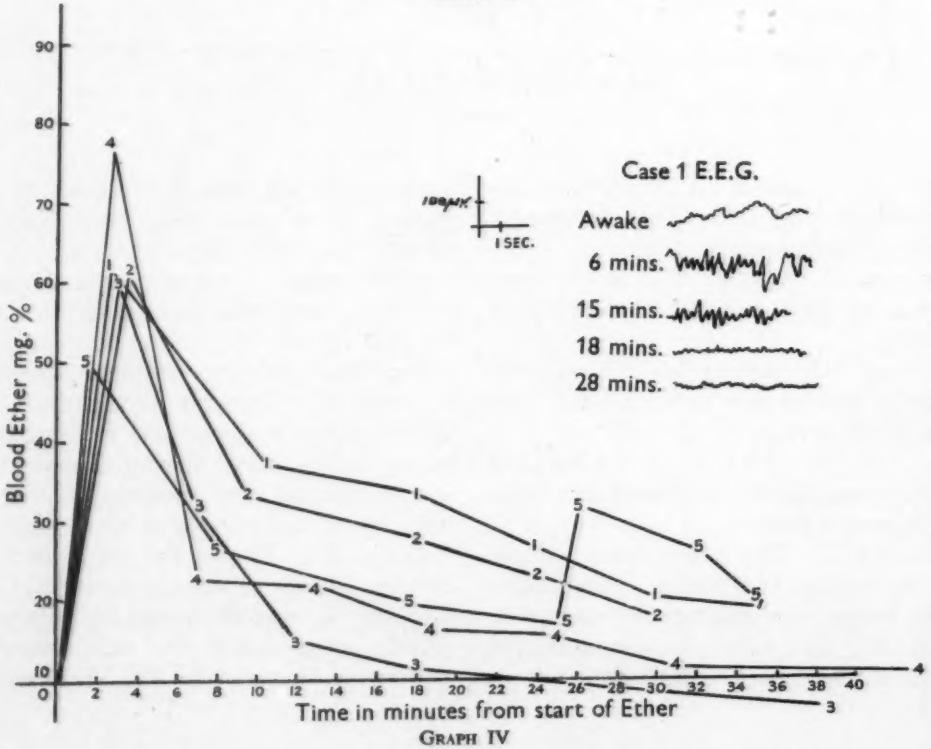
Graph IV. Group 'D'. Five cases lightly sedated and intubated and again consistent in pattern.

Graph V. This graph shows the plot of the average blood ether concentration over twenty-two minutes of Groups 'A' and 'B'. In Group 'A', the unsedated, non-intubated series, the curve shows a slower rise during induction followed by a gradual fall in blood ether level during maintenance. The curve of Group 'B', however, shows a very steep rise during induction; intubation was carried out at the peak of the curve and at only a little higher blood ether level than that attained in Group 'A'. Thereafter, the curve falls steeply during maintenance, the concentration falling below that of Group 'A' at nine minutes, and remaining substantially lower until the end of operation.

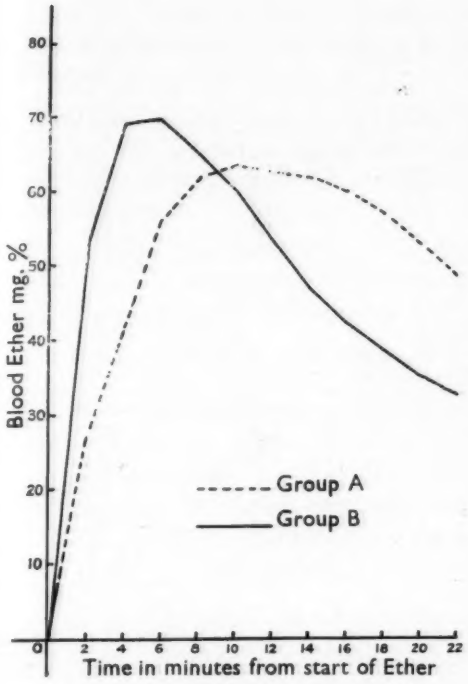
Graph VI. This is the graph of the average blood ether concentration of Groups 'C' and 'D' over 22 minutes. These curves follow the same general pattern as Groups 'A' and 'B' except



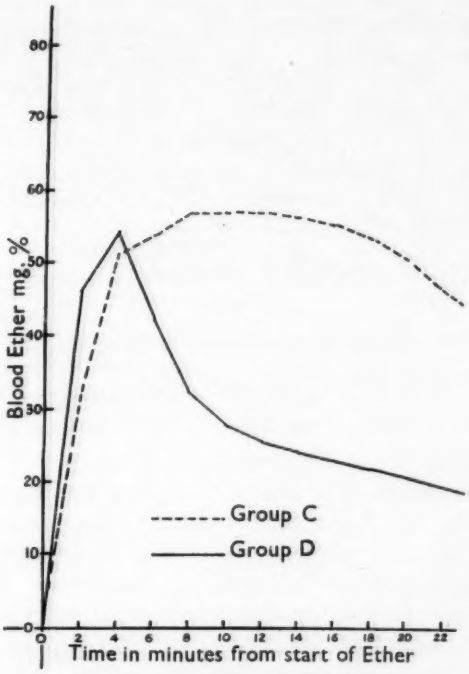
GRAPH III



GRAPH IV



GRAPH V



GRAPH VI

that in the sedated and intubated series—Group 'D', both intubation and maintenance could be carried out on a lower blood-ether concentration, presumably due to the depressant effect of the pethidine or morphine on the laryngeal and tracheal reflexes. A comparison of Groups 'A' and 'C' shows that sedation did not materially affect the blood ether level required to achieve a given plane as judged by the classical signs of anaesthesia. This has already been shown by Calderone (1935).

We felt that these findings were of some importance in operative obstetrics and therefore applied the same technique to a number of Caesarean sections. Maternal blood samples were taken at the time of intubation or "settling" and again as the umbilical cord was clamped. Samples of blood were then taken from the vein and an artery of a section of the cord between clamps, within one or two minutes of delivery. The maternal blood ether levels were essentially similar to those in the corresponding groups undergoing minor surgical procedures (tables I and II).

The blood ether concentrations which we found in the umbilical cord are consistently lower than those previously reported as causing foetal depression, particularly in those cases which were intubated. The average of our results was 27.6 mg./100 ml. Smith and Barker

TABLE I

Non-intubated cases

Time from start of ether to reach stage III(i) 4½ to 6½ minutes. Time from start of ether to clamping cord 10 to 12½ minutes.

	Maternal blood ether mg./100 ml.		Umbilical cord blood ether mg./100 ml.		
	Stage III(i)	Cord clamped	Artery	Vein	Sedation within 6 hours
1	53.0	38.0	20.0	31.0	No
2	70.0	45.0	22.0	37.0	No
3	53.0	55.0	35.0	49.0	Yes
4	55.0	55.0	—	34.0	Yes
				Average: 37.7	

TABLE II

Intubated cases

Time from start of ether to reach stage III(i) 1½ to 2½ minutes. Time from start of ether to clamping cord 7 to 14 minutes.

	Maternal blood ether mg./100 ml.		Umbilical cord blood ether mg./100 ml.		
	Stage III(i)	Cord clamped	Artery	Vein	Sedation within 6 hours
1	82.0	—	—	15.0	No
2	56.0	12.0	8.0	11.0	Yes
3	71.0	21.0	10.0	19.0	No
4	79.0	30.0	22.0	25.0	Yes
				Average: 17.5	

(1942) found that the onset of respiration was delayed when the ether level in the umbilical vein was 79.3 mg. per cent (18 estimations) and that resuscitation was required when the umbilical vein level reached 102.8 mg. per cent (8 estimations), and that in 50 estimations where the infant breathed at once, the average ether level in the umbilical vein was 64.3 mg. per cent.

Intubation, particularly in emergency cases, may be a desirable safeguard but the decision against it is frequently taken because of the fear that unduly deep anaesthesia would be needed for the act of intubation and that this might lead to foetal distress.

Our results suggest that the average concentration of ether in the maternal blood throughout the operation may be expected to be lower, if intubation be carried out, since anaesthesia can be lightened much more freely when a tube is in place. The peak concentration at the moment of intubation need be little if any higher than the concentration needed to maintain tranquil anaesthesia for extraperitoneal surgery, without a tube, provided that a suitable induction technique be used. The concentration in the maternal blood 10 to 20 minutes after induction, that is at about the time of extraction of the foetus, may be expected to be substantially lower with an endotracheal tube than without.

SUMMARY

The blood ether concentrations attained with two techniques of nitrous oxide, oxygen and diethyl ether anaesthesia are compared with the following results:

1. When carbon dioxide is used as a respiratory stimulant deep anaesthesia is not required for oral intubation.

2. With a cuffed endotracheal tube in place, anaesthesia can be maintained with lower blood ether levels than if the patient is not intubated.

3. Sedation has little effect on the blood ether concentration required to achieve a given depth of anaesthesia.

4. These findings hold good for obstetric anaesthesia, and with these techniques low blood ether levels in the foetus are obtained.

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BOOK REVIEW

Medical Electronics. By G. E. Donovan. Published by Butterworth & Co., Ltd. Pp. 200; 30 illustrations. Price £1 10*s*.

Many doctors interested in electronics must have considered the possibility of writing a review of present-day electronic practices in medicine, but have been daunted by the complexity of the task. Dr. Donovan has made the attempt with a considerable measure of success.

From his preface one can have no doubt that the author was well aware of the difficulties in attempting to "provide a bird's eye view of the uses of electronics in medicine . . . though omitting all mathematical considerations and circuit diagrams."

The scope of the book is very wide. It ranges from the fundamental biological effects of radiations through laboratory instruments for the measurement of biological quantities, to an extensive review of electronic instruments which assist clinical observation.

When so much is attempted, it is easy to find points of criticism. Of course there are omissions, and the book is uneven in emphasis. Readers who need the explanations of the first chapters are likely to be a good deal puzzled by some of the electronic terms freely used later on. But, again remembering the size of the job, it is surprising that the unevenness of stress is not more marked and that the omissions are so few. A book of this sort is necessarily written quickly, for the scene changes so rapidly.

Non-technical readers seeking general information must be content to read the book "lightly" and when they do not comprehend, pass on without too much travail. They will learn much in this way, though they will not learn all.

Readers seeking to find out "how to do it" will be less satisfied, though the references at the end of each chapter give some introduction into the literature on each topic.

Dr. Donovan has tackled a job which badly needed doing and has given us a helpful book for which this reviewer is grateful.

Brit. J. Anaesth. (1954), **26**, 118.

ANNOTATION

THE VENOUS RETURN

DR. PRIMROSE'S paper draws our attention first to reduced venous return as a cause of circulatory failure. Since the total venous return in a minute is the cardiac output, a considerable reduction in cardiac output must occur, with the attendant consequences, and with the "efforts at compensation" recognized in physiology. Such cases of peripheral circulatory failure are observed in other fields, and may, it seems, occur in the course of anaesthesia, for it is implied that in other respects there was no reason to expect it in the four cases discussed.

The author's theory of the active participation of the venous system in the normal venous return is bound to create discussion. It is based fundamentally upon the view that mean aortic pressure is insufficient normally to return the cardiac output to the right side of the heart. Now, mean aortic pressure is able to do this, or it is not, and the argument seems to admit little alternative.

There may, perhaps, be no objection to putting it in a semi-quantitative way: suppose mean femoral arterial pressure to be 100 mm. Hg and femoral venous pressure to be, say, 5 mm. Hg. Suppose also that in the active capillary bed, mean "arterial capillary" pressure in the limb is 25 mm. Hg. There is a considerable drop in pressure (75 mm. Hg) as minute flow passes through the arteriolar and pre-capillary resistances. Is a pressure of 25 mm. Hg able to express this volume from the capillaries up to the femoral vein with a loss of pressure of only 20 mm. Hg? In other words, are we surprised to find that resistance offered to arterial flow is some 3.5 times as great as resistance to venous return from the capillaries? Having regard to the structure of the vessels concerned it is, perhaps, not a surprising ratio. There is, after all, nothing essentially unlikely in the association of a large blood flow rate with a comparatively small pressure — provided resistance offered to flow is small.

This is seen experimentally in the profound vaso-dilatation following administration of the tetra-ammonium componds. Intra-arterial pressure records show very low aortic pressures which are, nevertheless, able to expel the cardiac output through the greatly reduced peripheral resistance. In these cases the pressure difference between the aorta and capillaries may be very small indeed.

It is the question of capillary pressures which must concern the reader. The author believes that active capillary and venular "pumping" takes place which, aided by valves, propels the venous return. Blood is prevented from returning to the arterial system by the "arteriolar resistance". Presumably, therefore, arterial capillary pressure is the force impressed as the blood is "squeezed" between the capillaries and the arterioles. Alternatively, what is the origin of capillary pressure? Is it not probable that it is the remainder of aortic pressure after traversing the arteriole resistance? If it be so,

and a positive capillary pressure of aortic origin is agreed, then it cannot be self-evident that the pressure itself will not return blood adequately from the tissues. There must be evidence of a resistance of sufficient magnitude to make it impossible, or at least extremely improbable. The notable work of Pappenheimer and Soto-Rivera (1948) lends considerable support to the view that, in the hindlimb at least, it is the difference between femoral arterial and femoral venous pressures which determines blood flow rates through the limb. A rise of equal value in each of these pressures is found to make no difference to the flow rate of blood, despite a rise in capillary and venule pressure.

The active participation or otherwise of the capillaries and venules is one thing; their potential capacity as a blood reservoir is another. It is recognized and may be generally agreed that large volumes of blood may be "stored" by these vessels, and so lost to circulation. In this way venous return becomes increasingly less and cardiac output inevitably follows.

Finally, we must also ask what positive evidence exists which cannot be explained by straightforward "pressure-flow-resistance" principles. Space does not permit a lengthy discussion of all these interesting points raised in the paper, and one must suffice, namely the finding of Evans (1944) in respect to the adrenaline drip technique in high spinal anaesthesia. In this case adrenaline appears to be used primarily at least to maintain vasomotor tone and blood pressure. Evans does not publish the diastolic pressures of his subjects, but the systolic pressures appear to be within the normal range. This does not mean that adrenaline may not also serve to limit the capacity of the "venous reservoir", but Evans's results do not seem to suggest arterial pressures incapable of disposing of the cardiac output and its effective return.

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Brit. J. Anaesth. (1954), **26**, 120.

PNEUMOTHORAX COMPLICATING BRACHIAL PLEXUS BLOCK ANAESTHESIA

BY

HUGH Y. WISHART

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PNEUMOTHORAX complicating brachial plexus block is one of the known hazards of the technique. The standard textbooks of anaesthetics mention the complication and suggest that it is of infrequent occurrence and of little importance. A review of the literature available to the author confirms its infrequency. Bonica, Moore and Orlov (1949) reported its occurrence on nine occasions in 1,000 blocks. The patients were not distressed and all cleared up in a few days. Bonica and Moore (1950) reported 10 pneumothoraces in a series of 1,512 blocks, and again no patients were distressed except for slight pain on deep inspiration. All recovered within a few days without sequelae. Griswold and Woodson (1943) in a series of 100 blocks had 2 cases of pneumothorax, one being of 25 per cent, and both cleared up without trouble. Patrick (1940) had no cases in a series of 44 blocks, but suggested that the complication must occur. Macintosh and Mushin (1944) in a series of over 400 blocks had 3 pneumothoraces, none of which was serious.

Although the papers mentioned above confirm its infrequency and would appear to confirm its lack of significance when it does happen, Kulenkampff and Persky (1928) and Rhone (1935) both mention a case reported by Capelle (1916) of pneumothorax and severe surgical emphysema following puncture of the lung during brachial plexus block. This patient died as a result of the accident.

The production of a pneumothorax during brachial plexus block is unlikely to be caused by the temporary lodgement of the needle point in the pleural space, particularly when a fine needle is used. Macintosh and Mushin (1944) suggest that the pneumothorax is caused by actual puncture of lung tissue. The small valvular hole so produced results in air entering the pleural cavity on inspiration, with sealing on expiration when the lung collapses. The same authors suggest that minor degrees of pneumothorax may be missed if they occur without obvious symptoms.

Kulenkampff and Persky (1928) drew attention to the fact that an emphysematous lung apex is closer to the brachial plexus than is normal and is more liable to injury during brachial plexus block.

The following two cases, the first of which occurred in the author's own practice, would appear to be of interest as they illustrate some of the points noted above.

CASE 1

On the evening of 9.9.53 a male patient was admitted with a compound fracture of the proxi-

mal phalanx of the middle finger of the right hand. He was to be treated as an out-patient, anaesthesia by brachial plexus block was chosen and this was performed in the following way. The mid-point of the right clavicle was determined, a 3-inch 22 S.W.G. needle was introduced one centimetre above this point just lateral to the external jugular vein, and directed towards the third dorsal vertebra.

The needle failed to contact the first rib and after two or three attempts was re-introduced about one centimetre medial to the external jugular vein. The first rib was located and paraesthesiae elicited. Thirty-five ml. of 2 per cent procaine with 1 in 200,000 adrenaline were injected and a satisfactory block of the right brachial plexus produced. The operation was completed and the patient allowed to return home, having made no complaint during the induction of analgesia or later.

On reporting next morning for a penicillin injection (given routinely to all compound fracture cases) the patient mentioned casually that he had had a small haemoptysis during the night. He had experienced no other respiratory complaint and was not distressed in any way when examined. There was no surgical emphysema at the site of injection. An X-ray (fig. 1) showed a right pneumothorax of about 20 per cent with some collapse of right lung and a slight shift of the heart to the left; there was a small pleural effusion in the right costo-phrenic angle.

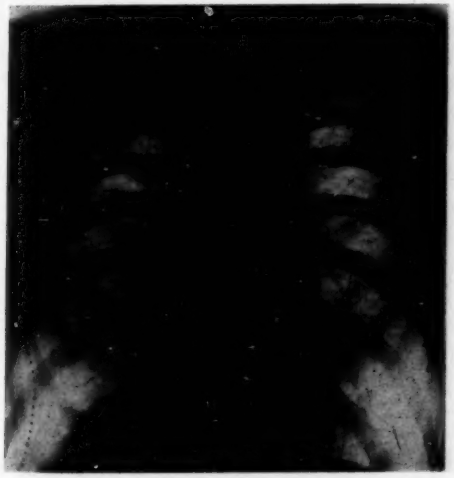

FIG. 1
(The pneumothorax has been outlined.)

As the patient lacked any symptom referable to his pneumothorax and was to receive a course of penicillin, it was decided to let him remain ambulant and to attend daily for review.

Four days later (14.9.53), an X-ray (fig. 2) showed a slight increase of the pneumothorax, the mediastinum still displaced to the left and the pleural effusion had not increased. The patient felt well and was symptom free. The course of penicillin was stopped—1,000,000 units initially followed by 450,000 units daily for 5 days.

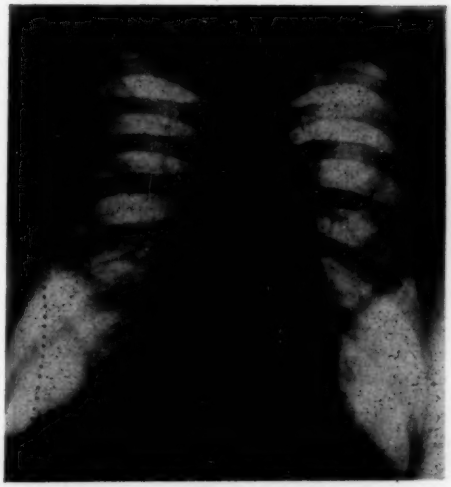

FIG. 2
(The pneumothorax has been outlined.)

The patient continued well and a third X-ray (30.9.53) (fig. 3) showed complete re-expansion of the right lung with resolution of the pleural effusion, the mediastinum now being central.

CASE 2

A male patient, aged 56 years, was admitted with Dupuytren's Contracture of the right hand. He was suffering from silicosis and chronic emphysema of both lungs. As he was orthopnoeic and was regarded as unfit for general anaesthesia, brachial plexus block was chosen. This was performed on 22.1.53 and shortly afterwards he became considerably distressed by

breathlessness and the operation was abandoned. The patient was returned to bed and was nursed sitting up and given general medical treatment, but his condition, although temporarily improved in the next few days, deteriorated markedly on 27.1.53. On 28.1.53 an X-ray showed a right pneumothorax with collapse of the right lung. General medical treatment was continued till 7.2.53 without improvement in his condition. To relieve the tension pneumothorax a needle was introduced into the pleural space and 900 ml. of air and some effusion were aspirated. The lung almost completely re-expanded and his symptoms were largely relieved. On 16.2.53 it became evident that there was a right basal pneumonia with secondary cardiac failure. With penicillin and medical treatment he gradually recovered and was discharged on 26.2.53.

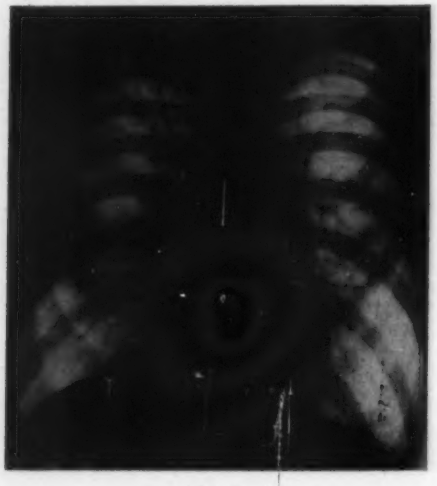

FIG. 3

DISCUSSION

The pneumothorax complicating Case 1 would have been missed but for the slight haemoptysis. This patient at no time complained of any other respiratory symptom, nor did he complain of pain when the anaesthesia was induced and the pneumothorax presumably occurred. The haemoptysis indicates that lung tissue was traumatized and the fact that the pneumothorax increased slightly during the first 4–5 days suggests that the valvular mechanism described by Macintosh and Mushin (1944) was responsible. Sealing of the lung puncture eventually limited the escape of air into the pleural cavity, reabsorption commenced and was complete within 3 weeks without sequelae.

In Case 2 the consequences of the lung puncture were much more serious. This patient had chronic emphysema of both lungs (itself a good indication for the employment of brachial plexus block) and it is likely that an emphysematous bulla was punctured. Such an injury would not seal off as quickly as it would in a normal lung, and as a result this patient suffered from a severe tension pneumothorax with marked respiratory and cardiac embarrassment. The prolonged collapse of the right lung in this case certainly favoured the onset of the pneumonic process which developed later.

In Case 1 the decision was made to treat the patient as an out-patient under surveillance as he showed no respiratory embarrassment. It was thought that his being ambulant would contribute to the re-expansion of the collapsed lung without recourse to physiotherapy and/or withdrawal of the air in the pleural space. The penicillin given for the compound fracture was considered sufficient cover for the small pleural effusion.

CONCLUSIONS

Most pneumothoraces complicating brachial plexus block would appear to clear up quickly without sequelae,

but should be watched closely till re-expansion of the collapsed lung is complete.

Special care should be exercised in performing brachial plexus block on patients suffering from chronic emphysema of the lungs on account of proximity of the emphysematous lung to the plexus with the possibility of an emphysematous bulla being punctured, an accident which might give rise to a tension pneumothorax. If a tension pneumothorax develops prompt surgical intervention is indicated to reduce the pressure and allow re-expansion of the collapsed lung, so relieving the respiratory and cardiac embarrassment. Re-expansion of the lung should also help to prevent the development of a pneumonic process in the collapsed lobes.

ACKNOWLEDGMENTS

My thanks are due to Mr. Traquair, Orthopaedic Surgeon to Hairmyres Hospital, for permission to publish the details of Case 2. I am indebted to Dr. H. H. Pinkerton for much help and useful criticism in the preparation of this paper.

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WORLD FEDERATION OF SOCIETIES OF ANAESTHESIOLOGISTS

THE first Congress of the World Federation of Societies of Anaesthesiologists will take place at Scheveningen near The Hague in September 1955. A provisional programme will be distributed in the course of 1954. The Organization Committee consists of:

Dr. C. B. Ritsema van Eck, Noorderbinnensingel 140[a], Groningen (*President*).

M. Mauve, Johannes Vermeerstraat 18, Heemstede (*Hon. Secretary*).

Dr. L. A. Boere, Laan van Oud-Poelgeest 7, Oegstgeest near Leyden (*Vice-President and Hon. Treasurer*).

A. M. Laterveer, Poggenbeekstraat 1, Arnhem.

J. van 't Oever, Schieweg 253[b], Rotterdam.

Dr. D. W. Swygman, Cronjestraat 1, Leeuwarden.

B. de Vries Robles, Haringvliestraat 17, Amsterdam Z.

Executive Manager: W. A. Fentener van Vlissingen.

Postal address: World Congress of Anaesthesiologists 1955, Noord-Houdringelaan 24, Bilthoven, Holland.

Brit. J. Anaesth. (1954), **26**, 124.

MODIFICATION OF A THREE-WAY TAP

BY

E. HARVEY FRANKS

Royal Air Force, Cardiff

INCREASING use is made of intravenous infusions for the administration of drugs during operation, e.g. adrenaline (Evans, 1944), Arfonad (Sadove, Wyant and Gleave, 1953), pethidine (Johnson, 1951), procaine, suxamethonium, and thiopentone/gallamine (Evans and Gray, 1953). If a blood transfusion becomes necessary there is the inconvenience of setting up another drip during the operation.

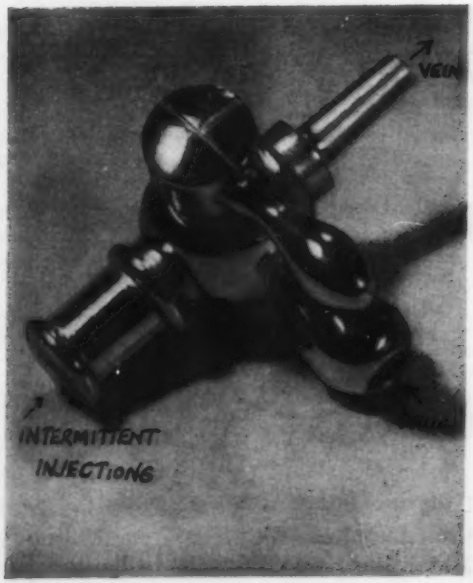

FIG. 1

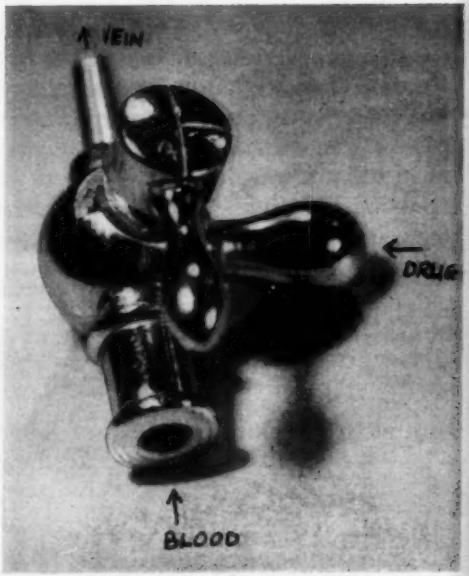

FIG. 2

Modification of the usual pattern three-way tap by filing down the metal stop-pin on the piston enables the apparatus to be used as shown in figure 2, as well as in the accepted way of figure 1. When a blood transfusion is needed all that is required is to insert the male connection of another giving-set into the three-way tap and to turn the piston into the position shown in figure 2.

Independent drip-rates at least up to 100 drips per minute for both the drug and blood are obtainable. The modified tap, which is easily sterilized by boiling, can be made in a few minutes from the usual piece of apparatus to be found in most anaesthetic departments.

I wish to thank Maurice Kidd, M.A., for the photographs.

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BOOK REVIEW

The Preparation and Writing of Medical Papers for Publication. By W. R. Bett. Published by Menley & James Ltd. Pp. 23.

A copy of the above has been received for review, and at the outset we would most sincerely recommend all our readers, and particularly our prospective contributors, to obtain, read and inwardly digest its contents.

It does not appear to be generally appreciated that there is more to writing a medical paper than just reporting a series of observations and conclusions. Those who have to edit papers which are submitted to journals know the labour that is involved in arranging headings and subheadings, not infrequently correcting spelling and punctuation, substituting official for proprietary names, substituting or adding metric dosages for the outmoded Apothecaries scale, and always arranging references according to the convention adopted by the particular journal.

In this invaluable little booklet will be found a discussion of the bibliographical tools of the intending author—how he will trace his references; the lists that are complementary to the well-known Index Medicus; where he will find simply explained the ordinary conventions of proof-correcting; all this, and a host of other information. Not many authors are aware that illustrations for papers should be prepared in a special way, and not too often can the importance of statistics be re-emphasized to medical authors. The pithy good sense that characterizes these invaluable twenty-three pages is exemplified by "The essence of style is the avoidance of (1) wind, (2) obscurity."

BOOK REVIEW

Ferdinand Sauerbruch: A Surgeon's Life. Translated by Fernand G. Renier and Anne Cliff. Published by Andre Deutsch. Pp. 297; 8 plates. Price 15*s*.

This is a fascinating autobiography of Sauerbruch, which has been very well translated into English.

An early experience in treating a penetrating sword wound of the lung turned his attention to chest surgery. His first chief, von Mikulicz, constantly reminded his young men of the scourge of pulmonary tuberculosis which challenged the surgeon's art. Sauerbruch experimented on dogs in his negative pressure chamber, but at first he failed to convince von Mikulicz of its value.

But soon von Mikulicz was converted, and together they demonstrated the chamber to the Berlin Surgical Congress. Unfortunately when von Mikulicz did the first thoracotomy on a woman with cancer of the gullet, the pressure valves of the chamber failed and the patient died when the pleural cavity was opened. But the next operation on a woman with a retrosternal tumour was entirely successful. The chest was now another field open to the surgeon. Sauerbruch's fame was assured.

The story and excitement of those days is graphically told. Then follows the promotion of the author to surgical chairs in the minor Universities of Greifswald and Marburg; in 1910 he was called to Zurich, his first important chair. Here he developed lung surgery, for the sanitoria of Switzerland were quick to provide him with patients.

His private practice quickly grew, including such patients as the wealthy Baron Rothschild and the temporarily exiled and impecunious King Constantine of Greece. During the first World War, Sauerbruch acted as secret courier between the Kaiser and the Sultan of Turkey. In the summer of 1918 he went to Munich, and was condemned to death and nearly shot by communists during the rebellion. With peace came recognition of him throughout the world. His last move, in 1927, was to the Charité in Berlin.

The concluding chapters deal with the death of Hindenburg, whom Sauerbruch attended in his last illness, and with the advent of the Third Reich. It is evident that Sauerbruch was not a Nazi—he was never interested in politics. On the contrary, he was friendly with General Beck and was lucky not to be arrested in connection with the July plot against Hitler in 1944.

The end of the War saw the author amputating limbs in the basement of the Charité, living on biscuits and Scotch whisky, till the Russians arrived.

The author did not suffer fools gladly —constantly we read that he lost his temper when confronted with stupidity or opposition. His assistants feared him; they had every reason to do so. It is a book to be recommended for the medical man as well as for the layman.

FACULTY NEWS

DR. H. J. V. MORTON has been nominated to give the next Frederic Hewitt Lecture in the Autumn of 1954, and Dr. W. W. Mushin to give the Joseph Clover Lecture in March 1955.

THE following are the names of the eligible Fellows who are candidates for a seat on the Board of the Faculty of Anaesthetists at an election to be held on Wednesday, March 10, 1954. There are three vacancies.

Robert William Cope
Frankis Tilney Evans
Richard Penn Harbord
Ronald Jarman
Edith Joan Millar
Hugh John Vivian Morton
William Woolf Mushin
Herbert Henry Pinkerton
Reginald Ernest Pleasance
Archibald Herbert Galley
George Leslie Feneley
Arthur Ivor Parry Brown
Stanley Arthur Mason
James Frederick Bereen
Gordon Sheridan Alfred Knowles

BEGINNING on March 25, the Faculty are beginning another three-week Anaesthetics Course which will include 40 lectures, clinical work three mornings a week, and tutorials each evening. Fees will be £15 15*s*. for lectures, £15 15*s*. for tutorials, and £5 5*s*. for the clinical work. The following is the programme:

FIRST WEEK

Thursday, March 25

10.0 –11.0 | Physical Problems in Anaesthesia.
11.30–12.30 | Prof. E. A. Pask
2.0 – 3.0 Functional Anatomy of the Air Passages. Dr. C. F. Scurr
3.30– 4.30 Tests of Respiratory Function and the Treatment of Respiratory Paralysis. Dr. B. A. Sellick
5.0 – 6.0 Tutorials.

Friday, March 26

9.0 –12.0 Hospital Visits.
2.0 – 3.0 The Modification of Respiration by Pathological and Clinical Factors. Dr. A. I. Parry Brown
3.30– 4.30 Respiration during Clinical Anaesthesia. Dr. H. J. V. Morton
5.0 – 6.0 Tutorials.

Monday, March 29

10.0 –11.0 | Anaesthesia for Thoracic Surgery.
11.30–12.30 | Dr. H. J. V. Morton
2.0 – 3.0 Explosions in the Operating Theatre. Dr. H. G. Epstein
3.30– 4.30 Anaesthesia for Special Investigations. Dr. H. G. B. Lucas
5.0 – 6.0 Tutorials.

Tuesday, March 30

9.0 –12.0 Hospital Visits.
2.0 – 3.0 Clinical Implications of Hypotensive Techniques. Dr. J. B. Wyman
3.30– 4.30 Developmental Abnormalities of the Cardiovascular and Respiratory Systems. Mr. N. Barrett
5.0 – 6.0 Tutorials.

Wednesday, March 31

9.0 –12.0 Hospital Visits.
2.0 – 3.0 Effects of General Anaesthesia on Cardiovascular Function. Dr. R. P. Harbord
3.30– 4.30 Anaesthesia for Cardiovascular Surgery. Dr. E. H. Rink
5.0 – 6.0 Tutorials.

SECOND WEEK

Thursday, April 1

10.0 –11.0 Assessment of Cardiovascular Disease. Prof. A. Kekwick
11.30–12.30 | Some Specific Nerve Blocks:
2.0 – 3.0 | Their Anatomy and Techniques. Dr. W. W. Mushin
3.30– 4.30 Anaesthesia and Analgesia in Obstetrics. Dr. W. D. Wylie
5.0 – 6.0 Tutorials.

Friday, April 2

9.0 –12.0 Hospital Visits.
2.0 – 3.0 Spinal Anaesthesia. Dr. Frankis T. Evans
3.30– 4.30 Epidural Analgesia. Dr. P. R. Bromage
5.0 – 6.0 Tutorials.

Monday, April 5

9.30–10.30 Dental Anaesthesia in Adults and Children. Dr. W. S. McConnell
11.0 –12.0 } The Use of Intravenous Fluids.
2.0 – 3.0 } Mr. H. E. Lockhart Mummery
3.30– 4.30 }
5.0 – 6.0 Tutorials.

Tuesday, April 6

9.0 –12.0 Hospital Visits.
2.0 – 3.0 General Considerations of the Pharmacology of the Local Analgesics. Prof. C. A. Keele
3.30– 4.30 Diagnostic and Therapeutic Applications of the Local Analgesics. Dr. A. H. Galley
5.0 – 6.0 Tutorials.

Wednesday, April 7

9.0 –12.0 Hospital Visits.
2.0 – 3.0 } Anaesthesia for Children.
3.30– 4.30 } Dr. G. Jackson Rees
5.0 – 6.0 Tutorials.

THIRD WEEK

Thursday, April 8

9.30–10.30 } Transmission and Block of Nerve
11.0 –12.0 } Impulses. Prof. W. D. M. Paton
2.0 – 3.0 Muscle Relaxants in Anaesthetic Practice. Dr. T. Cecil Gray
3.30– 4.30 Temperature Control during Anaesthesia. Dr. T. Cecil Gray
5.0 – 6.0 Tutorials.

Friday, April 9

9.0 –12.0 Hospital Visits.
2.0 – 3.0 Analgesic Drugs. Dr. R. F. Woolmer
3.30– 4.30 Special Problems of Pre- and Post-Operative Care. Dr. W. D. Wylie
5.0 – 6.0 Tutorials.

Monday, April 12

9.30–10.30 Anaesthesia in Relation to Normal and Abnormal Endocrine Activity. Dr. W. D. Wylie
11.0 –12.0 Anaesthesia in Relation to Normal and Abnormal Endocrine Activity. Dr. H. C. Churchill Davidson
3.30– 4.30 Problems of Neurosurgical Anaesthesia. Dr. A. J. H. Hewer
5.0 – 6.0 Tutorials.

Tuesday, April 13

9.0 –12.0 Hospital Visits.
2.0 – 3.0 The Analeptic Drugs and their Clinical Application. Prof. C. A. Keele

Tuesday, April 13 (continued)

3.30– 4.30 Renal and Hepatic Function in Relation to Anaesthesia. Dr. H. C. Churchill Davidson
5.0 – 6.0 Tutorials.

Wednesday, April 14

9.0 –12.0 Hospital Visits.
Radiological Interpretation.

A COURSE of approximately 20 lectures in Pharmacology will be held at the College from May 3 to May 14 inclusive, at a fee of £6 6*s*. Details and application forms for these Courses can be obtained from the Secretary of the College.

THE British Council and the Faculty of Anaesthetists are again co-operating in a Short Course for fifteen overseas students, mostly surgeons, and this will take place from March 15 to March 26 inclusive. The students will spend their time visiting London Hospitals, in groups of five, to watch anaesthetic techniques and administration. The Faculty are arranging the details with the anaesthetists at the Hospitals concerned, and the British Council are organizing the accommodadation and transport, etc., of those concerned.

FIFTEEN successful candidates have been awarded the Fellowship as a result of the first examination held in December 1953. They are:

Sidebotham, Robert Noel
Thompson, Peter Wentworth
Adams, Adam Stanislaw
Christie, Thomas Hildred
Bhattacharjee, Birendralall
Bryson, James Robertson
Bhojraj, Yeshwant Ganesh
Chadwick, David Airey
Lomaz, Jozef
Mesham, Paul Ronald
Vanniasingham, Thomas Wethavanm
Carr, Dorothy Agnes Mary
Clarke, James Camac
Cook, Vernon Bruce
Atkinson, Richard Stuart

THE following have been elected to the Fellowship of the Faculty:

Adams, B. W.
Alberts, F.
Anderson, J. T.
Anson, B. J. B.
Aukin, W.
Badman, G. E.
Bain, J. W. L.
Bannister, A. R.
Bilsland, W. L.
Bliss, R. P.
Bloom, N. H.
Bodington, J. R.
Boyle, M. M.
Braddon, I. G.
Brown, A.
Brown, D. J. A.
Butt, A. N. G.
Clayton, R. B.
Comerford, R. E.
Conroy, P. B.
Corcoran, C. J.
Corfield, H. S. A.
Cough, D. H.
Crawford, J.
Culbert, T. D.
Dangerfield, D.
Danks, S.
Davies, D.
Denton, M. V. H.
Dixon, J. H.
Domaingue, F. G.
Donaldson, G. G.
Ennis, G. E.
Evans, G. Lavery
Franklin, H.
Friedland, C. M.
Fairlie, H.
Gifford, L. A.
Gibson, J. B.
Godwin, E.
Goodhew, D. J. H.
Gotla, D. W.
Gray, A. J.
Gusterson, F. R.
Hamilton, J.
Hardie, A. W.
Harrison, K. C.
Harvey, Phoebe N.
Hill, F. W.
Holland, C. B.
Holloway, M. H. W.
Holms, A.
Houldsworth, H. B.
Hoult, E. A.
Howe, M. S.
Hughes, D. R.
Inglis, J. McN.
Inkster, J. S.
Jackson, H.
Jefferson, H.
Jenkins, D. C. R.
Johnston, N. C.
Keating, V. J.
Kirkpatrick, C.
Lancaster, F. M.
Leitch, A.
Lewis, C. B.
Lewis, J. K.
Lewis, J. W.
Little, B. R.
Lipton, S.
Lithman, L. H.
Lockett, J. M.
Lyall, D.
McCarthy, A.
Macdonagh, P. F. M.
McErvel, T.
McEwen, W. G.
Machell, E. S.
McMillan, R. L.
Mason, J. C.
Marshall, T.
Martin, L. V.
Mather, L.
Matheson, J. B.
Middleton, H. G.
Miles, T. F.
Munro, W. D.
Murtagh, G. P.
North, J.
Northover, P. T.
O'Connor, A.
Ofenheim, A.
O'Neill, M. B.
Parker, B. M.
Perredes, J. F.
Payne, J. P.
Pope, E. S.
Pollock, M. A.
Purves, G. F.
Raffan, A. W.
Reese, D. F.
Rhodes, E.
Richardson, H. J.
Rigg, D. R.
Ritchie, L. W.
Robertshaw, F. L.
Robinson, J. A. W.
Roussel, O. N.
Russell, F. R.
Ryan, A. R.
Scarr, J. B.
Schofield, J. E.
Seward, E. H.
Shannon, D. W.
Shaw, R.
Sherwood, M. P.
Sneddon, J.
Steen, T. R.
Thomas, J. L.
Tyson, W. J.
Waine, T. E.
Walch, R. C.
Walker, H.
Walter, W. J.
Watson, M.
Warrick, J. W.
Westall, Una
Westhead, P.
Whitby, J. D.
Wiggins, W. R. D.
Williams, A. M.
Willis, Gertrude A.

PAST AND PRESENT MEMBERS OF THE FACULTY OF ANAESTHETISTS OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

Left to right: Drs. A. H. Musgrove, K. Lloyd-Williams, W. A. Low, A. D. Marston, Sir Cecil Wakeley, Bt. (President of the Royal College of Surgeons), Dr. Bernard Johnson (Dean of the Faculty of Anaesthetists), Dr. T. Cecil Gray (Vice-Dean of the Faculty of Anaesthetists), Drs. E. S. Rowbotham, J. H. T. Challis, G. Edwards, F. T. Evans, B. L. S. Murtagh, R. F. Woolmer, C. Langton Hewer, A. J. W. Beard, M. D. Nosworthy, H. C. Churchill-Davidson, R. E. Pleasance, Mr. W. F. Davis (Secretary), Drs. I. W. Magill, V. F. Hall, S. A. Mason, E. A. Pask, and G. S. W. Organe.

Educational Supplement

March 1954

REVIEW ARTICLE

MODERN AUTOMATIC RESPIRATORS

BY

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Welsh National School of Medicine, Cardiff

AND

L. RENDELL-BAKER

United Cardiff Hospitals and Welsh Regional Hospital Board

THE widespread use of controlled respiration* in anaesthesia, and the interest shown in the Copenhagen method of treating spino-bulbar poliomyelitis (Lassen, 1953) has created a demand for devices to take the place of the anaesthetist's hand. The demand is comparatively recent; the existence of such devices is not. Excellent ones had been constructed over 40 years ago (Pulmotor, 1911; Janeway, 1913); their present day descendants spring from the researches of Frenckner and Crafoord, whose efficient Spiropulsator was already in use in 1934 (Frenckner, 1934).

An automatic respirator can do little more than take the place of a skilled pair of hands rhythmically squeezing the reservoir bag of an anaesthetic apparatus. In designing an automatic respirator, therefore, the acceptable characteristics of skilled manual ventilation must be understood. The peak pressure required to inflate the chest of an apnoeic patient may be as high as 20 to 25 cm. H_2O. When the chest is opened and the only resistance to be overcome is the elasticity of the lungs, the pressure required is smaller, say 10 to 15 cm. H_2O. During intra-thoracic operations, when retractors hold the lung back, the pressure necessary to ensure adequate ventilation may again be as high as 20 to 25 cm. H_2O. The distension of the lungs at each inspiration to be considered adequate, depends on a number of factors. In general, at each breath the lung will probably be inflated to a point short of that in normal life. It need never be inflated so that it extends beyond the confines of the thoracic wall opening. The minute volume ventilation should be sufficient to ensure perfect oxygenation and effective carbon dioxide removal. The flow pattern of gas in the respiratory tract should conform as nearly as possible to that in normal life (fig. 1). Examples of

*It should not be forgotten that there are considerable differences between controlled and natural respiration. These and the points for and against controlled respiration are set out elsewhere (Mushin and Rendell-Baker, 1953).

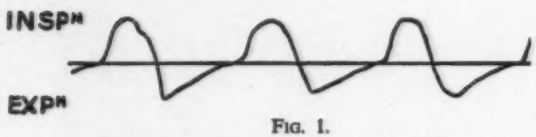

FIG. 1.
Respiratory flow pattern in spontaneous breathing (after Proctor and Hardy, 1949).

such patterns will be found in many papers (e.g., Fleisch, 1925; Maloney et al., 1953; Proctor and Hardy, 1949), although it is somewhat doubtful whether minor variations in the flow pattern are of great importance. Thus, the pattern produced by a simple piston pump, known as a "sine wave curve" (fig. 2), has so far not led to any demonstrable harm in patients ventilated with such a pump for long periods of time. The peak of inspiration should not be prolonged because at this point there may be interference both with the circulation through the lung and with the filling of the right side of the heart. If the peak is prolonged, both gas transfer and cardiac output may be seriously hindered.

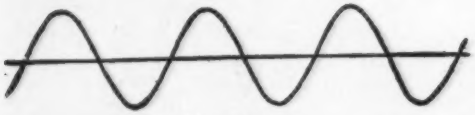

FIG. 2.
Flow pattern (sine wave) from pump respirator.

The rate of ventilation should be sufficiently rapid to give a good minute volume and good oxygenation, but not be so rapid that the reduction in cardiac output coincident with each ventilation peak becomes a serious complication. During manual ventilation with a thin rubber bag, excessive pressures which might injure the alveoli are unlikely, since the production of peak pressures in excess of say 30 cm. H_2O is very difficult in these circumstances. In the case of a mechanical device some safety valve must be included.

As soon as the peak pressure has been reached the patient's chest should be allowed to collapse at its own speed. This will only happen if the pressure is reduced rapidly to atmospheric. There is some evidence that a moderate negative pressure during the expiratory phase assists ventilation and counteracts the depression of cardiac output which quickly occurs during the peak pressure period of inspiration (Maloney et al., 1953).

All these desirable criteria must be met by a mechanical device. They may be set out thus:

A mechanical respirator:

(1) should reproduce as nearly as possible the normal pattern of respiratory gas flows, volume and pressure changes;

(2) should be sensitive to pressure changes within the chest so that "cycling" (i.e., the change from expiratory to inspiratory phase or vice versa) occurs at a pre-set peak pressure. Pump type mechanisms, described later, cannot do this. The pump delivers a certain volume before cycling, a pre-set blow-off valve being usually provided to prevent excessive pressures from being developed;

(3) should give visual and if possible audible evidence of (i) leaks, (ii) obstruction to the airway, (iii) mechanical failure of the machine;

(4) should be quiet in operation;

(5) should be reliable and robust;

(6) should be safe from fire or explosion.

INDICATIONS FOR THE USE OF AUTOMATIC RESPIRATORS

In our opinion no mechanical device can altogether take the place of sensitive

and skilled hands operating a reservoir bag. To suggest, as has been done, that a machine, however elaborate, " gives " the anaesthetic, is a point of view which cannot be accepted. Therefore while an automatic respirator *can* be used on any occasion on which controlled respiration is used, our own indications for its use are as follows:

(1) it sets free the hands of the anaesthetist working alone and enables him to perform other tasks connected with the administration of the anaesthetic;

(2) it relieves the physical fatigue of the anaesthetist's hands and arms during long cases;

(3) it relieves the fatigue of mind (or monotony) of squeezing the bag during such long cases;

(4) in circumstances in which the anaesthetist is not wholly skilled in manual ventilation a mechanical respirator provides the surgeon with an operating field which moves to a uniform rhythm, possibly facilitating certain delicate dissections, particularly within the thorax.

DANGERS

The use of an automatic respirator is not free from certain dangers:

1. The anaesthetist may be lulled into a false sense of security. He may assume that because the machine " clicks " regularly the patient is being adequately ventilated. On more than one occasion during our travels we have seen patients exhibiting obvious cyanosis and cardio-respiratory distress without exciting any concern on the part of the anaesthetist or the surgeon, because the machine connected to the patient was apparently working well.

2. Uninformed surgical opinion may take the view that a machine is all that is necessary for good chest anaesthesia, when in fact it can never be more than an adjunct to, and not a substitute for, an experienced anaesthetist.

3. The information which the anaesthetist gains from his handling of the bag is lost. Almost subconsciously the experienced anaesthetist compensates for the compression of the lung by surgical retractors. He quickly senses from the increased resistance to his efforts that further doses of relaxants are needed. The presence or otherwise of respiratory obstruction and accumulation of secretions are easily detected by the experienced hand. Every flicker of the diaphragm and every embryo cough is felt. By constantly watching the lungs the action of the hands is correlated to the effects produced inside the chest. None of these things is done by the mechanical respirator.

4. Mechanical failure may occur at any time and without warning. Unless such failure is quickly detected, and skilled manual ventilation substituted, the patient, if he remains apnoeic, may quickly die.

In considering the various respirators, we deal here only with those currently available, or those exhibiting particularly interesting features, but the ones described will give sufficient indication of the general structural types and variations. The ingenuity and efficiency of mechanical respirators described in the literature of a past generation has by no means been excelled and it is probable that the present demand will bring to light inventions for long buried in old papers. Inventors are well advised to study these papers.

TYPES OF RESPIRATORS

In general respirators may be classified into two groups. In the first, the basic mechanism is a pump by which a certain volume, sometimes fixed, sometimes variable, is delivered at each stroke of the pump. Naturally, the pressure developed in the chest will depend on such things as the speed with which the volume is delivered into the chest and on the size of the patient's chest. A safety valve of some sort is therefore incorporated so that a pre-set peak pressure is not exceeded.

In the second type, often called "pressure sensitive" respirators, cycling occurs when a pre-set pressure is reached within the chest, irrespective of the volume of gas which has been delivered into the lungs.

PUMP TYPE RESPIRATORS

In general, these suffer from the disadvantage that a fixed or pre-set volume is forced into the lungs irrespective of the pressure developed. A safety valve must therefore be incorporated. However, if the pressure to which the latter is set, is exceeded before the pump has completed its stroke, the pressure curve within the chest will be flat topped and the lungs will be held distended until the pump reaches the end of its stroke. It is therefore highly desirable that the stroke volume of such a pump should be capable of easy and rapid variation during the course of an anaesthetic. The early examples of the pump respirator are those of Pinson and Bryce (1944) and Mørch (1947)—the latter being notable for its variable stroke volume.

The two modern examples are those of James (1950) and Beaver (1953).

A. The James Respirator (Pacific Electric Co. Ltd., Melbourne, Australia).

In this apparatus (fig. 3) a concertina bag is compressed 20 times a minute by a sparkless electric motor, through a lever mechanism similar to the valve gear link motion used on some locomotives.

This link system permits rapid changes in the stroke volume to be made without stopping the electric motor.

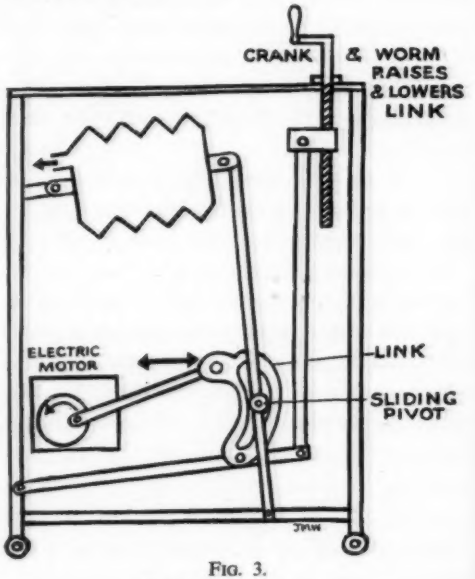

FIG. 3.
The James Respirator.

A water manometer indicates the pressures produced and also acts as a safety valve. The peak pressure is set by adjusting the water level in the manometer. To permit the escape of surplus gases when using nitrous oxide and oxygen mixtures, a spring-loaded spill-valve is provided and is set to blow off just before the manometer reaches peak pressure.

Figure 4 shows details of the ingenious manometer. By pushing the plunger down,

the blow-off pressure can be increased and is indicated by the water level. If this pressure is exceeded gas will bubble audibly and visibly through the water and escape through openings at the top.

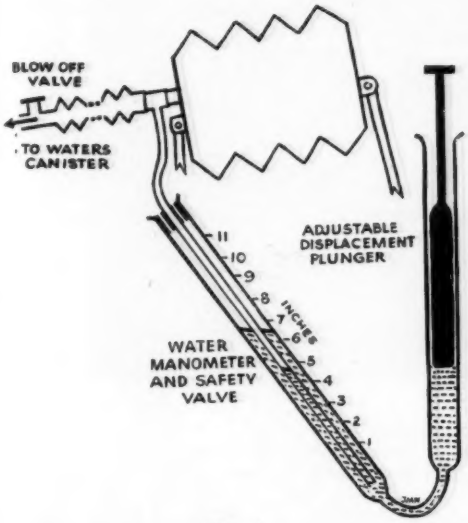

FIG. 4.

The James Respirator—manometer

In use, the stroke volume is adjusted to suit the patient's estimated needs. The manometer is set to a suitable blow-off pressure, say 20 cm. H_2O. Further adjustments might be made as the anaesthetic proceeds. The peak pressure will depend upon whether the chest is open or not, and upon the elasticity of the lungs. Obstruction of the airway or forcible retraction of the lung will be shown at once by the escape of gas through the spill valve, and through the water manometer.

This apparatus is a highly-developed pump-type respirator, but does not appear to be generally known in this country. Subsequent and similar machines, though with fewer refinements, have been described without reference to it.

*B. The Beaver Respirator** (British Oxygen Co. Ltd.).

In this apparatus (fig. 5) a small electric motor alternately compresses and expands a bellows by means of a crank drive. On the compression stroke the contents of the bellows are forced into the patient. On the expansion stroke the negative pressure created opens an expiratory valve allowing the exhalations to escape into the atmosphere. A separate valve acts as a safety blow-off and is adjusted to the peak pressure desired during inspiration. This pressure is indicated on a manometer. The rate of respiration is fixed at either 18 or 14 per minute. In the event of electrical breakdown the link mechanism is easily disconnected and the bellows worked by hand. The capacity of the bellows is 1400 ml.; no means of adjustment of the stroke volume is provided. In patients with small tidal capacities, the lungs may be held in distension through a large part of the inspiratory phase.

C. Radcliffe Respiration Pumps (H. G. East & Co., Oxford).

Russell and Schuster (1953) described two ingenious automatic electrically-operated respiration pumps, intended for use in bulbar poliomyelitis.

The first consists of an Oxford Inflating Bellows (Macintosh 1953) and an electric motor. During expiration the bellows and a known weight are lifted. During inspira-

* The machine described here was developed quickly in response to an urgent demand before an expected epidemic of poliomyelitis. We understand that considerable improvements are being incorporated in future models.

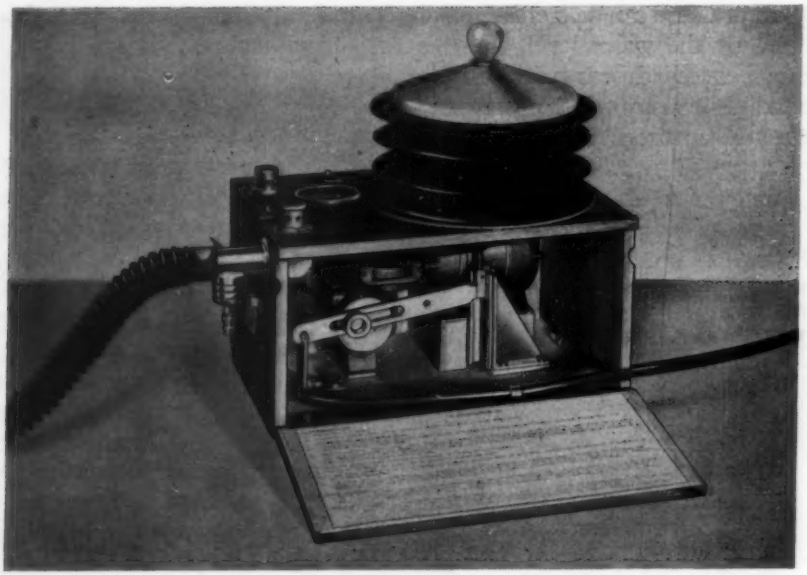

FIG. 5.
The Beaver Respirator.
The open box shows the electric motor, crank drive and actuating rod which passes through the centre of the bellows.

tion the weight alone compresses the bellows. By altering the size of the weight, both the rate of inspiration and the maximum pressure can be varied. By altering the speed of the motor the rate of respiration and the length of expiration is varied. The stroke volume can also be adjusted by a stop.

The other pattern utilizes the suction pipeline provided in many hospitals to raise a piston in a cylinder. When the piston falls, the patient's chest is inflated. An electric gramophone motor operates a rotating valve which connects the suction intermittently to the cylinder.

PRESSURE SENSITIVE RESPIRATORS

In these, a supply of compressed air either from a cylinder or from a compressor inflates the patient's chest. When a pre-set pressure is reached the supply is is interrupted and the intra-thoracic pressure allowed to fall quickly to atmospheric. The manometric device which effects the "cycling" consists, in some forms of apparatus, of a diaphragm, the movement of which actuates valves, in others of a water manometer or a bellows closing electrical contacts, actuating electrically-operated valves.

A. Aga Vivrator (Gasaccumulator Co., Stockholm, Sweden).

Until recently this apparatus was only available combined with the flow meters and absorption system of the Stille anaesthetic apparatus, the whole being known as the Spiropulsator. As such it was the

modern pioneer of mechanical respirators and deservedly achieved a world-wide reputation.

We have chosen to describe the Vivrator because it is the essential part of the Spiropulsator. It is now sold separately and since it can be connected to any anaesthetic apparatus, it is likely to be of interest to British anaesthetists.

The apparatus (fig. 6) consists of the pressure sensitive valve (2) and the pressure chamber (3). A supply of compressed air is attached at (1) to the pressure sensitive valve.

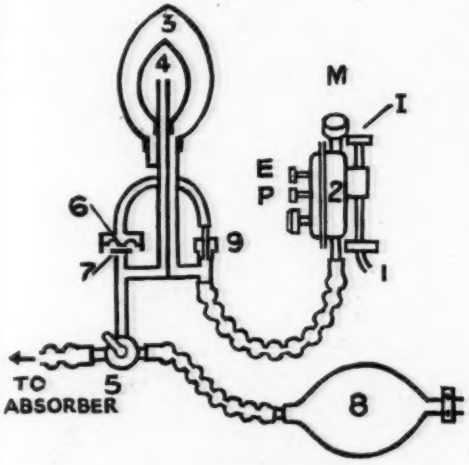

FIG. 6.
The Aga Vivrator.

I—Tap controlling compressed-air supply.
M—Manometer.
E—Tap controlling duration of expiration.
P—Tap controlling maximum pressure.
1—Compressed-air inlet.
2—Pressure sensitive valve.
3—Pressure chamber.
4—Reservoir bag.
5—Tap controlling manual bag.
6—Diaphragm controlling blow-off.
7—Excess gas blow-off.
8—Bag for manual operation.
9—Plug (removed to permit spontaneous respiration from bag).

Inspiratory phase. Compressed air flows through the valve along the corrugated tubing into the pressure chamber (3) forcing the gases from the reservoir bag (4) via tap (5) to the patient's lungs. Compressed air also passes along the narrow tube (9) to the diaphragm (6) which descends holding the spill valve (7) closed during inspiration.

Expiratory phase. When the selected pressure is reached, the magnetic control valve (2) cycles. The compressed air supply is cut off and the air in the pressure chamber (3) allowed to pass out into the atmosphere through the expiratory valve. The gases from the patient's lungs flow back into the reservoir bag (4), any excess of gas volume escaping through the spill valve (7). The reservoir bag (8) is provided for manual inflation, if desired.

The bag (4) takes the place of the normal reservoir bag of a closed circuit apparatus. The Vivrator can be put out of action temporarily by switching off the compressed air supply by means of the tap (I) and removing the plug (9). The pressure chamber is now in free communication with the atmosphere, and the patient can breathe freely from bag (4).

The details of the Vivrator pressure sensitive valve are shown in figure 7.

Compressed air enters at (1) and is controlled by the tap (I) which determines the length of inspiration. The compressed air enters the main chamber (2) and passes to the plastic compression chamber (fig. 6) via the corrugated tubing (3). While this is happening, the diaphragm (4) is gradually displaced, pulling with it the rod (5) to which are attached the valves. This rod moves between two magnets (6) which

cause it to flick quickly from one to the other, giving the valves (7) and (8) a positive action. When the diaphragm has reached a certain point, the rod (5) flicks over closing valve (8) and opening valve (7). The pressure in the patient's chest falls rapidly to atmospheric. The diaphragm now begins to return to its former position. The speed with which it does this, however, is controlled by the air inlet tap (E) which therefore controls the

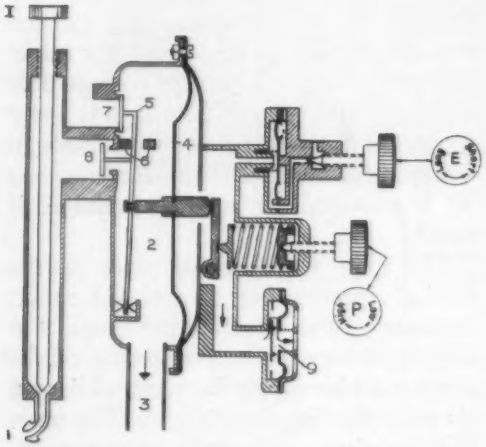

FIG. 7.
The Vivrator pressure sensitive valve.

length of expiration. When the diaphragm reaches a certain point the rod (5) again flicks over in the opposite direction, opening valve (8) and closing valve (7). The inspiratory phase then begins again. The pressure required to effect cycling is determined by the control (P) which by increasing the spring loading on the diaphragm, raises the pressure necessary to operate the valves (7) and (8). A one-way valve (9) allows the diaphragm to move easily during inspiration.

B. The Pulmopulsor (H. C. Ulrich, Ulm, Germany).

This German apparatus is similar in general design to the Vivrator. Here again the apparatus (fig. 8) is compact and the small unit can be attached to any closed circuit apparatus.

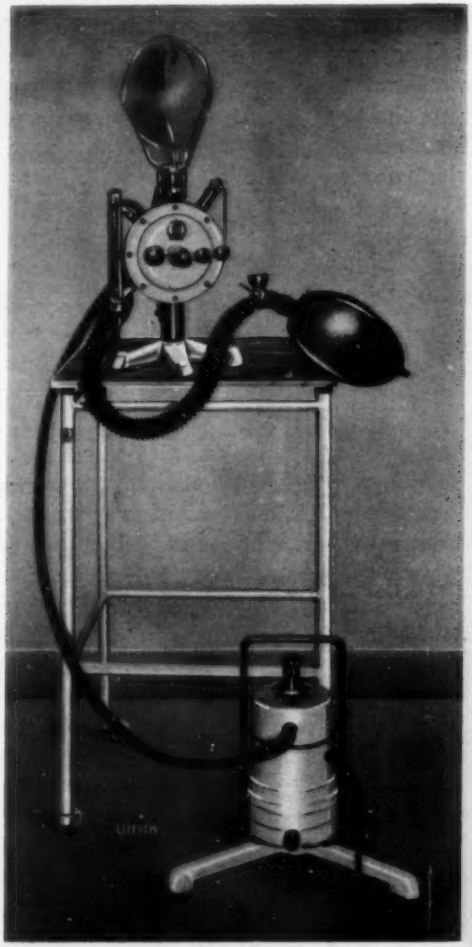

FIG. 8.
The Pulmopulsor.
The compressor stands on the floor. The neat and compact valve and pressure chamber takes up little room in the operating theatre.

C. The Blease Pulmoflator (Blease Equipment Co., London).

This apparatus (figs. 9 and 10) is another example of the diaphragm-operated type except that instead of magnets the flick-over mechanism is operated by a complex spring and ratchet device. It was a pioneer of apparatus of this kind in Britain and has deservedly achieved wide popularity.

Compressed gas enters the main chamber (A) from the compressor in the base of the instrument, and acts on the outside of the bellows (T) causing inspiration to occur and the patient's lungs to be inflated.

During this time, the diaphragm (D) is being displaced to the right until at a certain point the ratchet mechanism flicks over, opening valve (2) widely and allowing the pressure in the main chamber to fall quickly to atmospheric. Expiration is thus effected. The spring (E) pressing on the diaphragm causes it to begin to move in the opposite direction until the ratchet flicks over again, closing valve (2) and starting the inspiratory phase once more. The maximum pressure in the chest, shown on the manometer (P), is controlled by the lever (F) which alters the compression of the spring (E). The length of

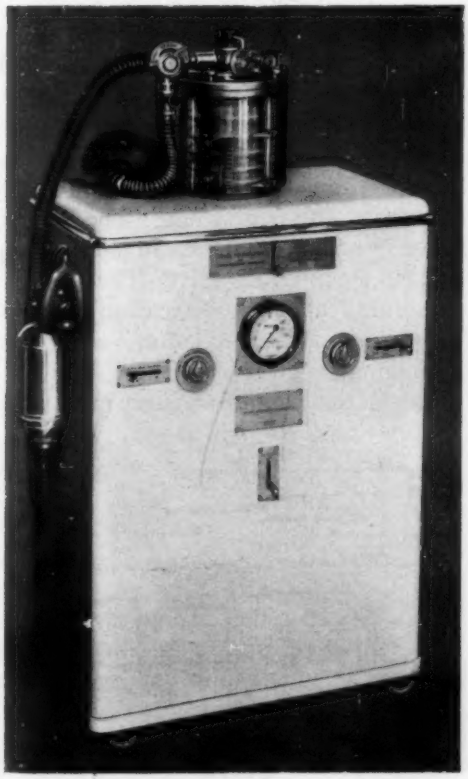

FIG. 9.
The Blease Pulmoflator.

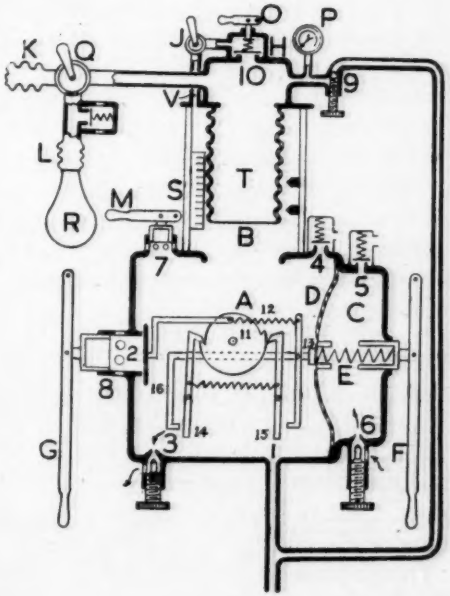

FIG. 10.
The Blease Pulmoflator—mechanism (Mushin and Rendell-Baker, 1953).

expiration is controlled by tap (6). The length of inspiration is controlled by tap (3). A scale (S) is provided so that the excursion of the bellows can be measured. Caution should be exercised before assuming that the volume of gas leaving the bellows is necessarily the volume entering the patient's lungs. This can only be so if leaks are rigorously excluded.

D. Williams Respirator.

This is a small compact device (fig. 11) which when plugged into the outlet from the Boyle's apparatus converts the continuous flow of gases into an intermittent

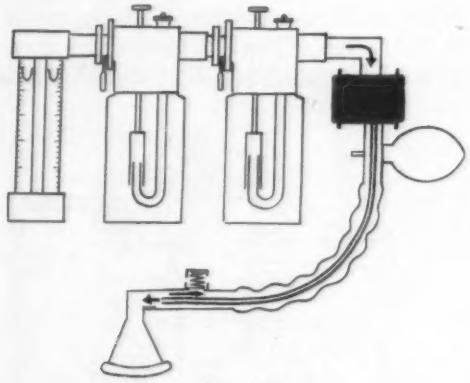

FIG. 11.
The Williams Respirator on Boyle's apparatus.

one. The apparatus (figs. 12 and 13) is provided with two stout rubber diaphragms (A) and (B) connected by a metal thrust rod (C). Lateral movement of this rod actuates the toggle trip switch (D) moving it from one extreme position to the other. The piston commutator of the gas valve (E) is attached to the trip switch.

In the inspiratory phase (fig. 12) gases flow from the anaesthetic apparatus, at the rate of 15 litres per minute, through the narrow tube (F) to inflate the patient's

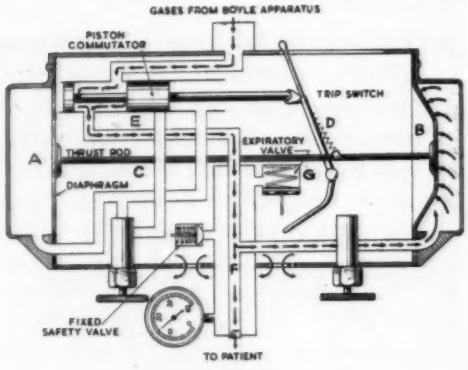

FIG. 12.
The Williams Respirator—mechanism—inspiratory phase.

lungs. Meanwhile the pressure exerted on diaphragm (B) gradually moves the thrust rod towards the left until the trip switch flicks over into the expiratory position.

In the expiratory phase (fig. 13) the gas supply to the patient is cut off by the valve (E) and the large expiratory valve (G) is opened by the trip switch. The expired gases return along the corrugated tubing and out through this valve. The circuit is thus unidirectional and the dead space for carbon dioxide accumulation is reduced to a minimum.

Inspiration commences once more when

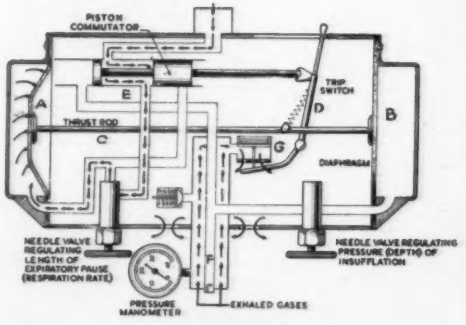

FIG. 13.
The Williams Respirator—mechanism—expiratory phase.

the gas passing through the left-hand needle valve has built up enough pressure on diaphragm (A) to reverse the position of the trip switch (D). The maximum pressure produced during inspiration and the duration of expiration is controlled by the two needle valves (fig. 13).

This respirator is the only one designed for use during anaesthesia without needing a closed circuit apparatus. Since soda lime is dispensed with, trichlorethylene may form part of the anaesthetic.

E. The Aintree Respirator (Medical & Industrial Equipment Co.).

Like the Spiropulsator, the Aintree Respirator (fig. 14) first described and designed by Esplen (1952) incorporates a magnet whose function, however, is rather

FIG. 14.
The Aintree Respirator.

different, as will be seen later. A special feature of this apparatus is that the compressed air inlet forms the jet of a venturi which provides a small amount of suction to assist the outflow of gases from the lungs during expiration.

Fig. 15 shows the magnetic control valve (11, 17 and 18) which regulates the supply of compressed air to the pressure chamber (1), compressing the reservoir bag (2) rhythmically. The water manometer (29) indicates the pressure and also acts as a safety blow-off valve.

A source of compressed air capable of supplying 30 litres per minute at 15 lb. per sq. in. (760 mm. Hg) pressure is attached to the apparatus at the inlet (7). The pressure is reduced to 2 lb. per sq. in. (100 mm. Hg) in the reducing valve (33). The tap (9) controls the flow of air passing through the venturi (8) into the chamber (10). The chamber is closed above by the disc valve (11), which is held down by the attraction between the magnet (18) and the metal plate (17). The air stream is diverted and flows both into the buffer bag (23) and into the pressure chamber (1). The pressure exerted on the reservoir bag (2) forces the gas mixture it contains into the patient's lungs until, at the pre-set pressure, the force of the magnet is overcome and valve (11) lifts, allowing the escape of the compressed air. The flow of compressed air through the venturi (8) produces a slight negative pressure in the pressure chamber (1) and this aids the flow of gases along the tubing from the patient, ensuring a rapid fall to atmospheric pressure in the patient's lungs. When the pressure under the valve (11) falls sufficiently, the attraction between the magnet and the metal plate becomes strong enough to close valve (11). The inspiratory phase then starts again. When tap (6) is open any excess of gas in the circuit can escape through the Kendrick valve (36).

Speed of Inspiration and Duration of Expiration. Tap (9) controls the speed of inspiration and the duration of the expira-

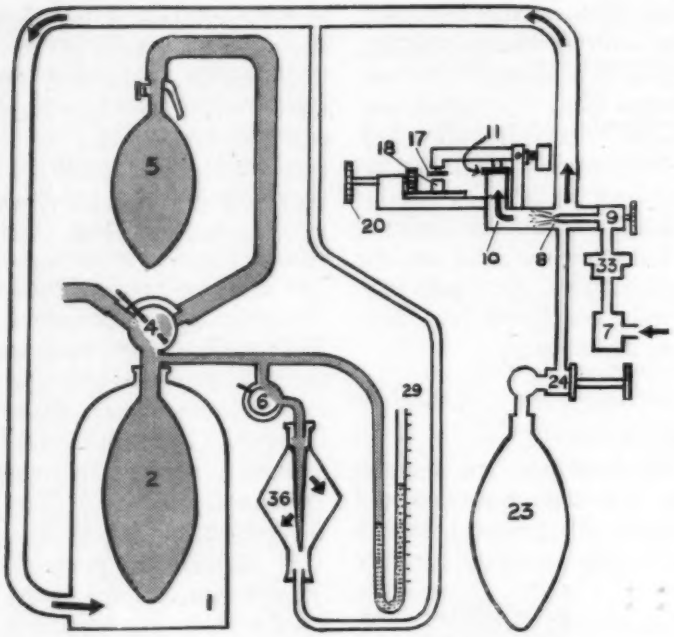

FIG. 15.
The Aintree Respirator—mechanism and circuit.

tory pause. The more compressed air admitted, the faster are the lungs inflated, and the greater is the pressure of compressed air holding valve (11) open during expiration. A longer expiratory pause results. A low flow of compressed air produces a slower inflation of the lungs and the valve (11) is not held open so long during expiration.

Frequency of Respiration. The bag (23) is intended to damp down extraneous pressure waves arising from, say handling of the lungs by the surgeon, which would otherwise cause the magnetic valve to "cycle" prematurely. With the valve (24) fully open the respiratory rate is 8 respirations per minute. As the valve is closed, reducing the capacity of the circuit, the respiratory rate increases up to a maximum of 30 per minute.

Pressure. The maximum pressure produced during inspiration is varied by knob (20), which raises or lowers the magnet (18). Raising the magnet closer to the metal plate (17) increases the loading on the valve (11). A higher pressure is therefore attained before the valve is lifted.

The Aintree Respirator is supplied with a separate compressor unit. This is intended to be kept outside the operating theatre, not only to reduce noise, but also because its motor and switch are not spark-proof. This would constitute an explosion risk in the presence of inflammable gases.

PRESSURE SENSITIVE RESPIRATORS WITH ELECTRICALLY-OPERATED VALVES

A. Bang Respirator (Bang, 1953).

This apparatus was originally designed during the Danish 1952 poliomyelitis epidemic, to provide continuous artificial respiration over long periods on patients with bulbo-spinal poliomyelitis. The apparatus differs from those already described, in that the gases, from the cylinder or compressor, motivating the apparatus, also enter the patient's lungs. The valve which controls the duration and depth of inspiration and the duration of expiration is controlled electrically.

Description (fig. 16). A cylinder of compressed air or oxygen mixture (A) is fitted with a contents gauge (B), a tap (C), and a flowmeter (D), Gas at the rate of 7–10 litres per minute is admitted to the circuit and distends the reservoir bag (E) which stores the gas during expiration and provides a low pressure (13–19 cm. H_2O) supply of gas for inflation of the patient's lungs.

Inspiration and expiration both take place through the valve (F). This consists of a piston fitting closely into a tube, round the ends of which are wound two solenoid coils (G). These, when energized, attract the metal piston to one or other end of the tube. The centre of the piston is cut away so that gas is able to flow through the inspiratory tube when the piston is in the left-hand position, and through the expiratory tube when it is in the right-hand position.

Two tubes, one for inspiration and one for expiration, pass from the valve and are joined together by a " Y " connection (H) near the patient. A side tube (K) from the expiratory tube passes to the manometer which therefore moves with the changes of pressure in the patient's lungs.

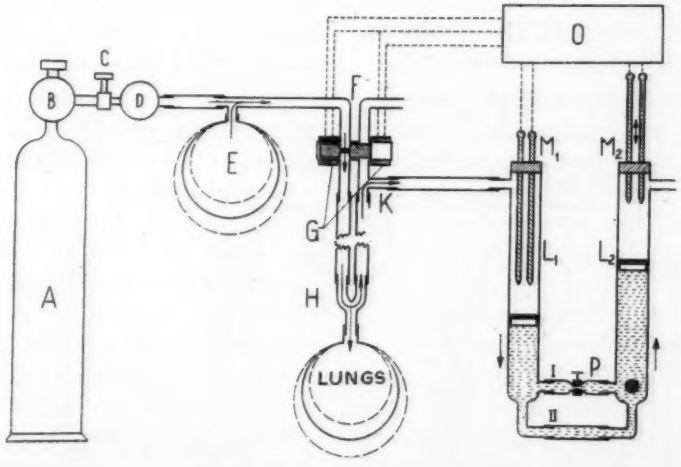

FIG. 16

The Bang Respirator (Bang, 1953).

The manometer consists of two tubes (L_1 and L_2), filled with an electrolyte solution, connected by two pieces of rubber tubing (I and II). Of these (I) can be partly occluded by the clamp (P). L_1 is airtight and connected to the patient's airway, whilst L_2 is open to the atmosphere and contains a light metal ball which allows liquid to enter by tube (II), but prevents egress by that route.

Thus, during inspiration the electrolyte flows rapidly into the right-hand tube L_2 as the pressure in the chest rises, but during expiration the fall of the electrolyte in the right-hand tube is hindered by the metal ball and the pinch-cock (P). Hence, the expiratory pause, after the pressure in the patient's airway has returned to atmospheric, can be varied by manipulation of the pinch-cock (P).

The electrodes (M_1) are adjusted so that contact with the electrolyte occurs at atmospheric pressure. These control the left-hand solenoid, which, when energized, initiates inspiration. The height of the electrodes (M_2) is varied to obtain the maximum inspiratory pressure desired.

The rate of respiration depends upon the speed of inspiration and the length of the expiratory pause. Inspiration can be speeded up by increasing the gas flow by opening tap (C). The expiratory pause is controlled by tap (P).

B. The Newcastle Respirator (Pask, 1953)

This apparatus (fig. 17), a modification of Bang's, utilizes an electrical circuit with relays and electrolytic contacts, to control the supply of compressed air or anaesthetic gases to the patient's lungs. It is thus pressure sensitive, but uses electrical power instead of gas pressure to activate directly or indirectly the various valves in the circuit.

The component parts of the apparatus are: a cylinder of compressed air (A) fitted with an Adams valve; a reservoir bag (B) which provides a supply of air at low pressure; a water safety blow-off valve (S.V.); a relay-controlled expiratory valve (E); a gas-actuated inspiratory valve (I); and a manometer filled with a solution of Na_2SO_4, containing the two sets of electrodes (C.

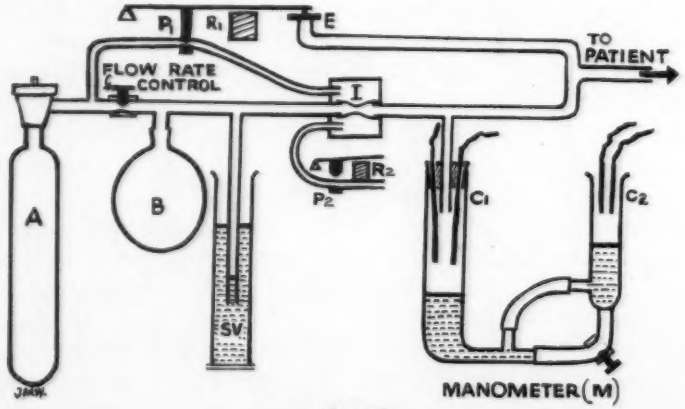

FIG. 17.
The Newcastle Respirator.

and C_2). The two electrical relays (R_1 and R_2) control pinch-cocks on the entry and exit tubes from the inspiratory valve chamber. By opening one and closing the other, the thin rubber tube of the inspiratory valve is either collapsed or patent.

Mode of operation. The electrical supply is switched on and the height of the electrodes (C_2) is adjusted to give the desired maximum inflation pressure. The flow of air from the cylinder (A) is set to the desired respiratory minute volume (about 10–12 litres/minute) and the depth of the tube in the water safety valve (S.V.) adjusted to about 50 cm. H_2O so that ample working pressure is available.

Inspiratory phase. Air flows at a steady rate from the cylinder (A), fills the reservoir bag (B), and passes through the inspiratory valve (I) to the patient's lungs. The relay (R_1) is held closed, both occluding the expiratory valve (E) and closing the pinch-cock (P_1).

As the pressure in the patient's lungs rises, the electrolyte level in the right-hand limb of the manometer (M) rises until it reaches the contacts (C_2).

Expiratory phase. When the circuit at C_2 is made, the actions of the two relays are reversed; (R_2) closes pinch-cock (P_2) and (R_1) opens the expiratory valve (E) and the pinch-cock (P_1). Air from the Adams valve then flows into the outer chamber of the inspiratory valve (I), causing the thin rubber tubing to collapse. The air from the patient's lungs flows out of the open expiratory valve (E).

The pressure in the lungs falls rapidly, and the electrolyte in the right-hand limb of the manometer falls until atmospheric pressure is reached, when the contacts (C_1) energize relay (R_1), closing the expiratory valve. The inspiratory cycle starts once more. The rate of fall of the electrolyte and with it the duration of the expiratory pause, can be controlled by the clamp on the rubber tubing connecting the two limbs of the manometer. The siting of the by-pass tube permits rapid response to increases of pressure, coupled with an adjustable delay in the response to falling pressure.

This apparatus was designed so that it could be constructed quickly and with a minimum of skill, in hospitals in time of epidemic. The source of electricity is a series of car batteries, though we have adapted the apparatus for A.C. mains by means of suitable relays and a rectifier transformer circuit. Our limited experience of this apparatus has demonstrated its reliability and efficiency, both in the operating theatre and for maintaining ventilation during prolonged apnoea as after craniotomy.

C. The Clevedon Respirator (Macrae et al., 1953).

This is another electrically-controlled apparatus based on, and very similar to, the Bang respirator. Like the Newcastle apparatus, it is designed to work from an accumulator and independently of the mains.

D. Mørch's Respirator.

The general principle of this is shown in figure 18, which is almost self-explanatory. The inspiratory and expiratory valves are operated by solenoids. These are actuated by a tilting mercury switch in contact with a pressure sensitive bellows.

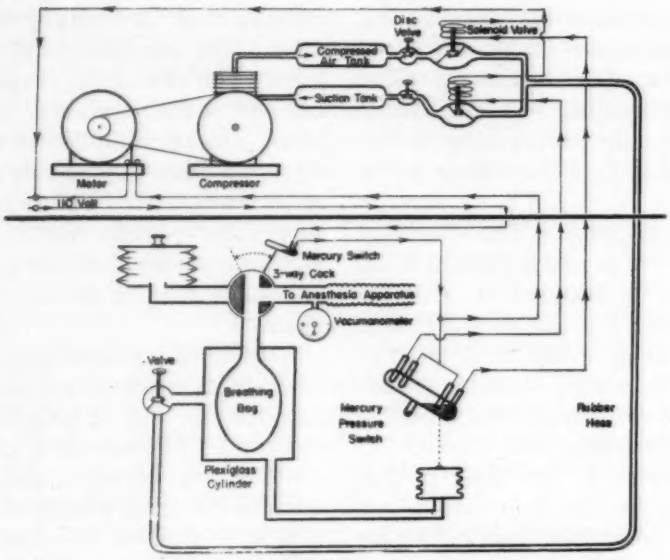

FIG. 18.
Mørch's Respirator (Mushin and Rendell-Baker, 1953).

CONCLUSION

Limited as we are by the confines of a journal, many other examples of automatic respirators have had to be omitted. Some of these are but modifications of those described, while others are still in the development stage or are not easily available in this country. Details of many of these will be found elsewhere (Mushin and Rendell-Baker, 1953).

The automatic respirator is no toy, but an undoubted boon to the anaesthetist and his patient. It has clearly come to stay, but if its benefits are to be realized, the machine must never be allowed to master the anaesthetist. That the automatic respirator is no more than a tool, albeit invaluable, is our present conviction. Well understood and wisely used it is another milestone in the march of anaesthetic progress.

ACKNOWLEDGMENTS

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A NEW DEPARTMENT OF ANAESTHETICS

BY

BERNARD JOHNSON

Dean of the Faculty of Anaesthetists
of the Royal College of Surgeons of England

THE post-graduate educational activities of the Faculty and the inauguration and management of an examination for Fellowship of the Faculty are now well known as Faculty achievements. The Board of Faculty felt, however, that since the Faculty is the national academic centre of British anaesthesia, it should play some part in research into the subject. Ideally, research is carried on by workers free from the responsibility of maintaining an anaesthetic service in a busy hospital and the instruction of undergraduates. Furthermore, it is essential that those engaged in research into a subject with such a wide field as anaesthesia need a close liaison and co-operation with departments of the basic sciences. All these facilities could be made available in the Royal College of Surgeons.

The Board of Faculty, therefore, put their case to the Council of the Royal College of Surgeons with the request that 3,000 sq. ft. of space be allocated in the new buildings of the College for the establishment of a Department of Anaesthetics.

The Council, of course, saw the need for such a department within the College, and instructed its Building Committee, under the chairmanship of Sir Stanford Cade, to consider the problem in con-

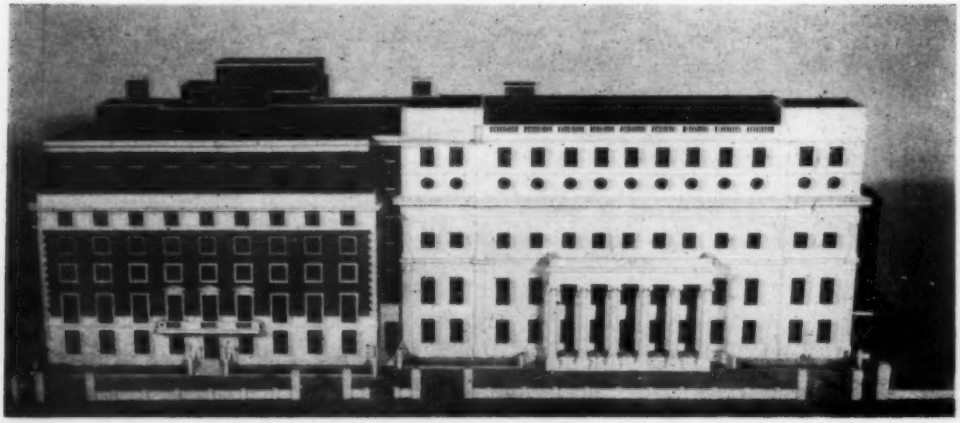

FIG. 1
The area in bold relief is the proposed Department of Anaesthetics.

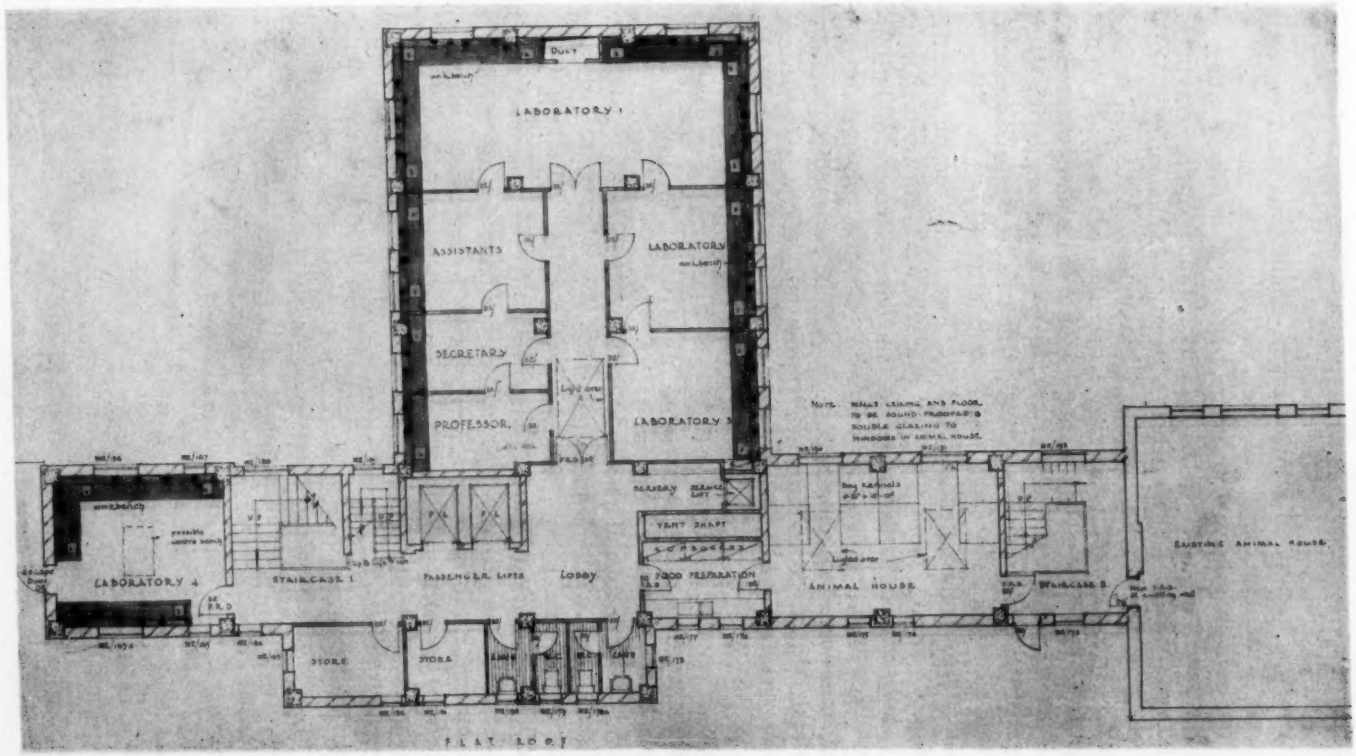
LABORATORY 1
ASSISTANTS
LABORATORY 2
SECRETARY
PROFESSOR
LABORATORY 3
LABORATORY 4
STAIRCASE 1
PASSENGER LIFTS
Lobby
FOOD PREPARATION
VENT SHAFT
ANIMAL HOUSE
STAIRCASE 2
EXISTING ANIMAL HOUSE
STORE
FLAT ROOF

FIG. 2

sultation with the Faculty and allocate suitable space. At that time, all the space in the new buildings had been allocated, but there was an area on the top floor of the new Nuffield College which, although set aside for another purpose, seemed ideally situated for this development (top left, fig. 1). The Building Committee decided that the need for a Department of Anaesthetics was so great that the whole of this area, some 3,600 sq. ft., should become the Department of Anaesthetics.

This, of course, was all that could be desired, for in this situation the new department would be in direct communication with the Department of Physiology, which is on the top floor of the main building.

The Council showed its determination to advance the science of anaesthesia by accepting this somewhat bold proposal of its Building Committee and by requesting the Faculty to consult immediately with the architect and submit detailed plans for the new Department. The Board of Faculty realized that, however carefully planned, no department can be made exactly as an unknown Director of that department would wish. Nevertheless, information was sought from all over the country and many research units were visited. With the resulting information, plans were drawn up which are likely to meet all the requirements of even the most exacting Director (fig. 2). Furthermore, the dividing walls will be so constructed that the size of rooms could be altered with a minimum of expense and labour. It is anticipated that the building of the Department will be completed about the middle of 1955.

Two things remain to be done. One is to select the staff for the Department, and the other—even more difficult—is to find the finance required to run it, some £12,000 per annum. It must be realized that the College, with the best will in the world, cannot possibly add this burden to its overstrained finances. It is up to us—the British anaesthetists—to support the vision of the College Council by finding the funds needed to establish a Research Unit in our academic centre. We must not only seize, but also seek opportunities to gain the interest of those who may be able to help. Here is an opportunity for all, medical and lay, to assist in a project which will help to establish for all time British anaesthesia in the leading position which it at present holds in the world.

Brit. J. Anaesth. (1954), **26**, 151.

REPRESENTATIVE BODIES

ASSOCIATION OF ANAESTHETISTS OF GREAT BRITAIN AND IRELAND

THE Association of Anaesthetists of Great Britain and Ireland was founded in 1932, under the Presidency of H. W. Featherstone, with the following objects:

(*a*) To promote the development of this branch of medicine.

(*b*) To co-ordinate the activities of anaesthetists.

(*c*) To represent anaesthetists and to protect their interests.

(*d*) To favour the establishment of a Diploma in Anaesthetics.

(*e*) To encourage friendship among anaesthetists. Ordinary Members were limited to one hundred and fifty in number, drawn from anaesthetists on the staffs of teaching hospitals or engaged in research.

While co-operating closely with the British Medical Association, from which it received invaluable assistance, the Association of Anaesthetists maintained an active and sturdy independence and soon became recognized as the authority on all matters concerning anaesthetists. The furtherance of its objects led it to the consideration of a wide variety of problems. These included such matters as the representation of anaesthetists on Medical Staff Committees, explosions in operating theatres, the administration of analgesia by midwives, payment of salaries to anaesthetists attached to Professorial Surgical Units, scales of fees paid by Local Authorities, fees for lecturers and examiners, nurse anaesthetists, the conduct of Coroners' Inquests into deaths associated with surgical operations and various details of anaesthetic apparatus. Then, as now, the Association has confined itself to problems peculiar to anaesthetists; those of a more general nature have been referred to bodies representative of the profession as a whole, usually the B.M.A.

Another object was attained with the institution, in 1935, of a Diploma in Anaesthetics, following negotiations with the Royal College of Surgeons and the Conjoint Board. This was a material contribution to the advancement in status of the specialty, and the Diploma soon became an essential requirement of candidates for posts at all important hospitals.

By 1943 it became evident that the restricted membership made it difficult for the Association to represent adequately the interests of the rapidly growing body of anaesthetists and the Constitution was amended accordingly. The existing Members became Fellows, and membership was thrown open to all those whose interests were predominantly in anaesthetics. There are now 229 Fellows and 696 Members. Fellows are elected annually from among the Members, not normally of less than seven years' standing.

There had long been felt a need for an official journal and, in 1945, the first issue of *Anaesthesia* was published, under the Editorship of C. Langton Hewer. This publishes Association and Faculty news, in addition to original articles, indices and abstracts, and has acquired international importance.

In 1949, as a result of the deliberations of a Joint Committee of the Royal College of Surgeons and the Association, was founded the Faculty of Anaesthetists within the College. The retiring President of the Association, A. D. Marston, became the first Dean of the Faculty. In this, the Association relinquished its academic functions which were, by now, too important to be carried on as a sideline of its activities; but it retained existing, and received further grants of money which have enabled it to support various forms of research by anaesthetists.

In the years before and immediately after the inception of the National Health Service the problems facing the Officers and Council became more anxious and urgent. Many of these have been successfully concluded, but others remain and new difficulties present themselves. Although the present position of anaesthetists is very greatly better than in 1932, the maintenance of the *status quo*, apart from any advancement, calls for constant vigilance which is best exercised by a body of anaesthetists, responsible to anaesthetists alone. It is the duty, as well as the privilege, of every anaesthetist to strengthen the work of the Association with his membership.

B.M.A. ANAESTHETISTS' GROUP

In 1946 it was clear that a closer connexion of anaesthetists with the work of the British Medical Association was desirable. Accordingly a Group of Anaesthetists was formed from members of both the Association of anaesthetists and the B.M.A. The decisions of the group are considered and may be supported by the central Consultants' and Specialists' Committee, on which it has one representative. From here recommendations are passed to the Joint Committee.

The problems which face the Group Committee are largely those of the Council of the Association of Anaesthetists, but any divergence of purpose is avoided by the fact that there is inevitably a considerable overlap in membership of the two bodies, and the Group Committee includes a co-opted member of the Council of the Association. Membership of the Group, without further subscription, is open to practising anaesthetists who are members of the B.M.A.

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